



جامعة صنعاء
كلية الطب والعلوم الصحية
دائرة العلوم السريرية الطبية
دفعة بصمه طبيب 31
طب بشري

OPHTHALMOLOGY



10 Ophthalmic LASER
16 Vitreous Humour
17 Glaucoma
18 Refractive Errors

part 3
Lectures

Lecture No.

Edited by: **خلدون المعمرى**

اللجنة العلمية Group C

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مندوب المجموعة

مسعد الصيادي

مع تحيات مكتبة الحزبي للخدمات الطلابية والمراجع الطبية
جولة الجامعة الجديدة تلفون 212863-777447225

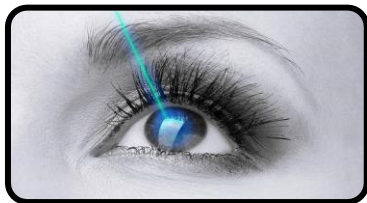
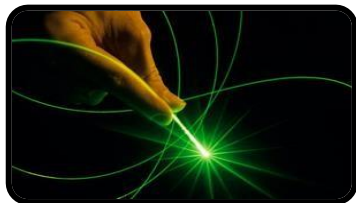
VISIT...

LANZAROTE
Caliente.COM

Ophthalmic LASER



Mahfouth A Bamashmus FRCSEd
FRCOphth Professor of Ophthalmology



What is LASER ?

L - Light

A - Amplification

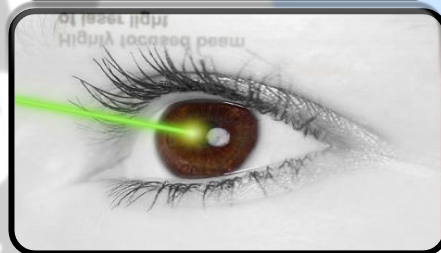
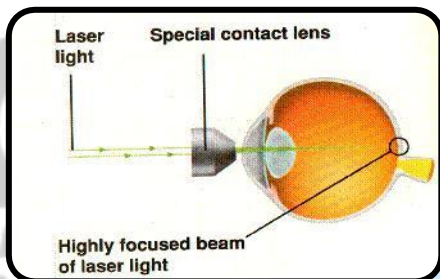
BY

S - Stimulated

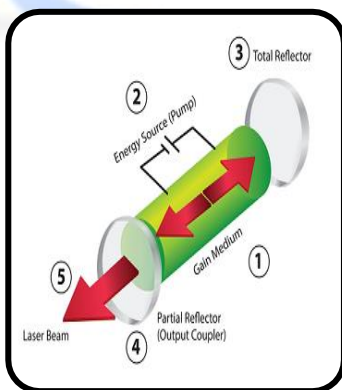
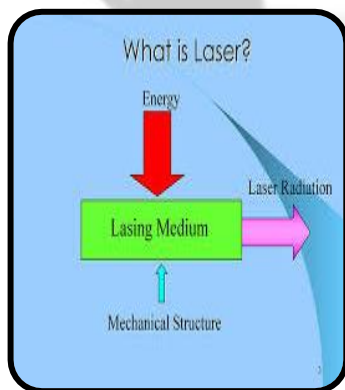
E - Emission

OF

R - Radiation



Substances have the property to “lase” i.e. absorb energy in one form & emit a new form of light energy which is more useful.

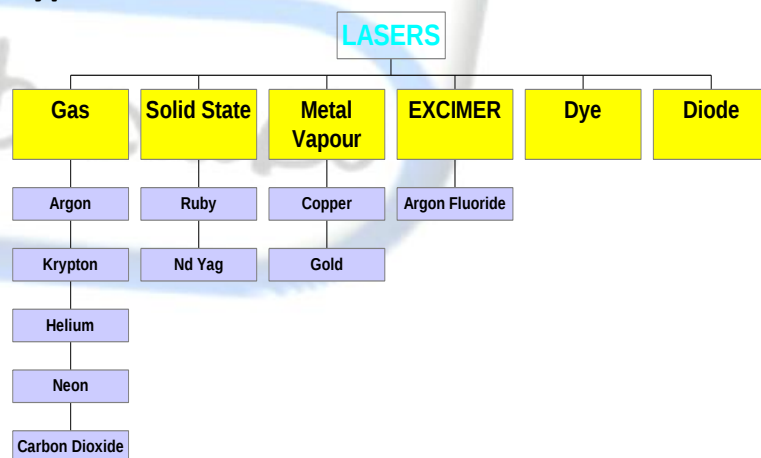


Why laser is particularly suitable for many medical applications?

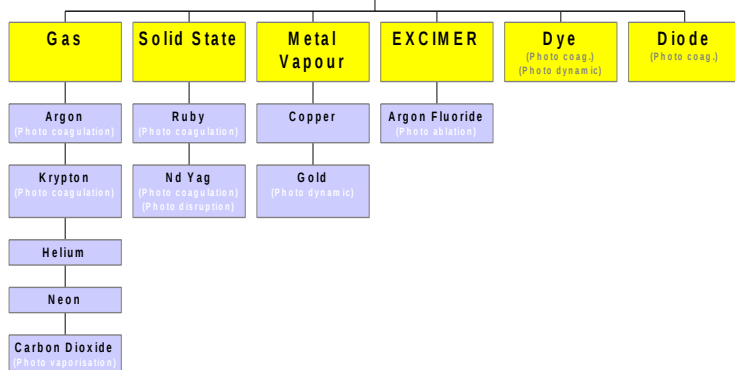
- Monochromaticity (only one wavelength that can be focused to a smaller spot than can white light)
- Directionality (narrow beam focusing light to a small spot)
- Coherence (propagated energy from the source is in phase and that improves focusing characteristics)
- Polarization (linear polarized light to allow maximum transmission without loss caused by reflection)
- Intensity (power in a beam of a given angular size {brightness per unit area})



Types of Lasers



LASERS



Radiation Wavelengths

- 193 nm - Excimer (Cornea)
- 488 - 514 nm - Argon (Retina)
- 780 - 840 nm - Diode
- 1064 nm - Nd Yag (Capsule)
- 10,600 nm - Carbon dioxide (Skin)

Lasers used in ophthalmology

Type	Atomic environment	Effect produced
Argon	Argon gas	photocoagulation
Krypton	Krypton gas	Photocoagulation
Diode	Diode crystal	Photocoagulation
Nd: YAG	A liquid dry or a solid compound of Yt-Al garnet+neodymium,	Photodisruption
Excimer	Helium and fluorine	Photoablation
Diode pumped Nd YAG	Diode and Nd: YAG crystal	Photocoagulation

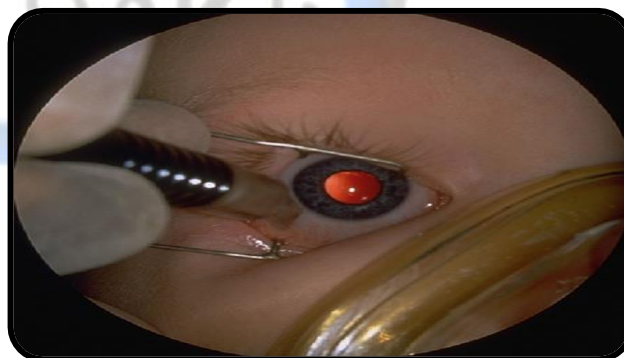
Delivery Systems

- Transpupillary - Slit lamp
- Transpupillary - Laser Indirect Ophthalmoscopy
- Trans scleral - Contact
- Trans scleral - Non contact
- Endophotocoagulation

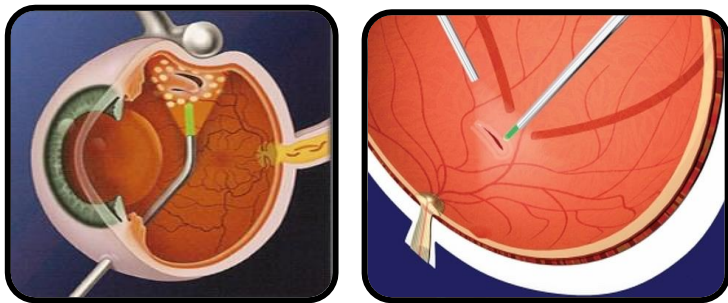
Delivery Systems- Slit lamp

Delivery Systems
Laser Indirect Ophthalmoscopy

Delivery Systems - Trans scleral



Delivery Systems - Endophotocoagulation



Uses

Diagnostic

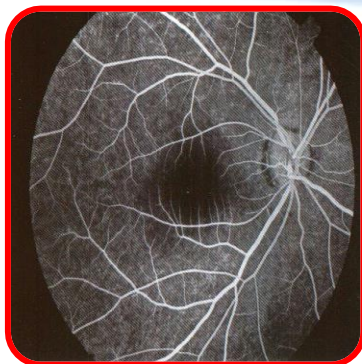
Therapeutic

Diagnostic Uses

1. Fundus Fluorescein Angiography
2. Laser Fluorescence Spectroscopy
3. Scanning Laser Ophthalmoscopy
4. Laser Interferometry

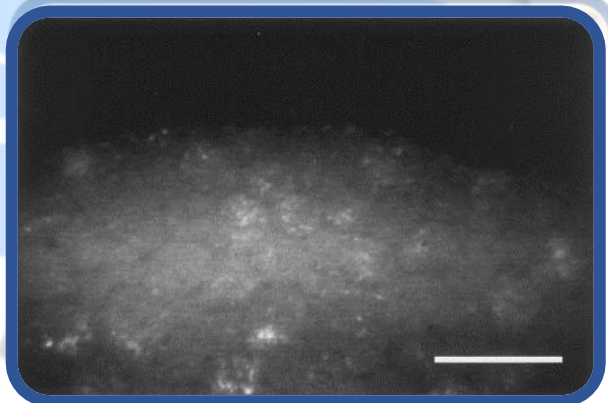
Diagnostic Uses

Fundus Fluorescein Angiography



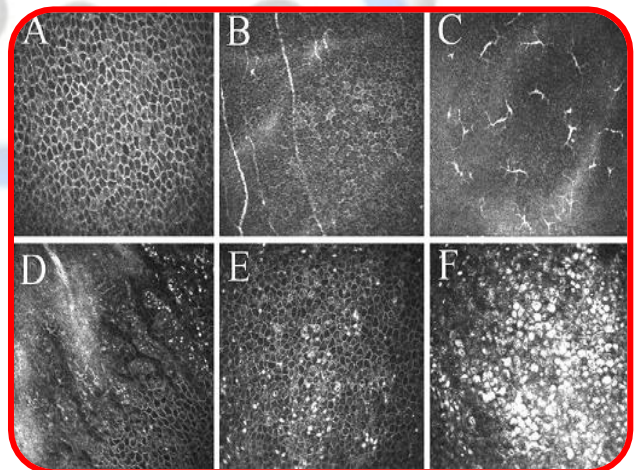
Diagnostic Uses

Scanning Laser Ophthalmoscopy



Diagnostic Uses

In VIVO Confocal Microscopy



Diagnostic Uses Laser Interferometry



Therapeutic Uses

Used For :-

1. Extra-ocular adnexae
2. Anterior Segment
3. Posterior Segment

Therapeutic Uses

Extraocular Adnexae

Therapeutic Uses Removal of lid masses



Laser Blepharoplasty (Smoothen Wrinkles)



Laser Blepharoplasty (Smoothen Wrinkles)



Laser Blepharoplasty (Smoothen Wrinkles)



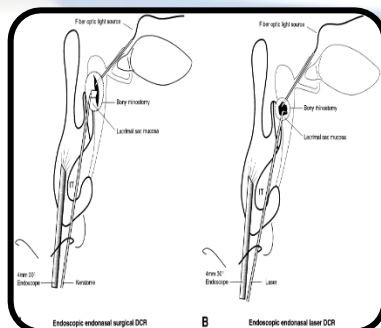
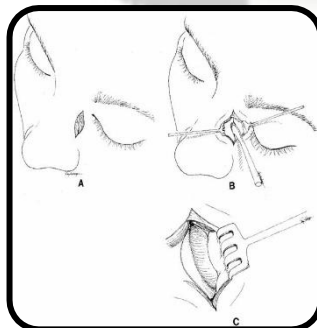
Laser For Skin Pigmentation



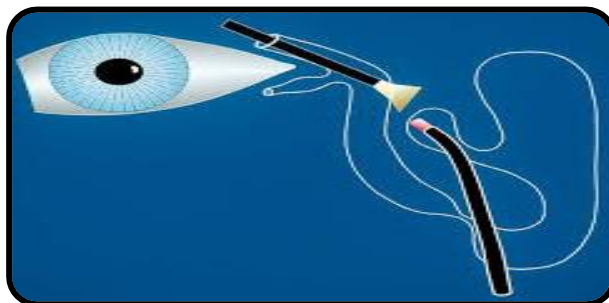
Laser For Skin Pigmentation



Therapeutic Uses Endonasal DCR



Therapeutic Uses - Endonasal DCR



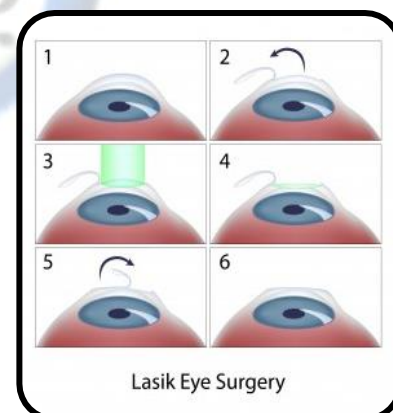
Therapeutic Uses Anterior Segment LASIK & Epi-LASIK (PRK)



Therapeutic Uses

What is LASIK & PRK ?

- Suitable for Myopia, Hypermetropia & Astigmatism
- 5 minutes per eye



Lasik Eye Surgery

Therapeutic Uses -LASIK



Suction Ring

Microkeratome

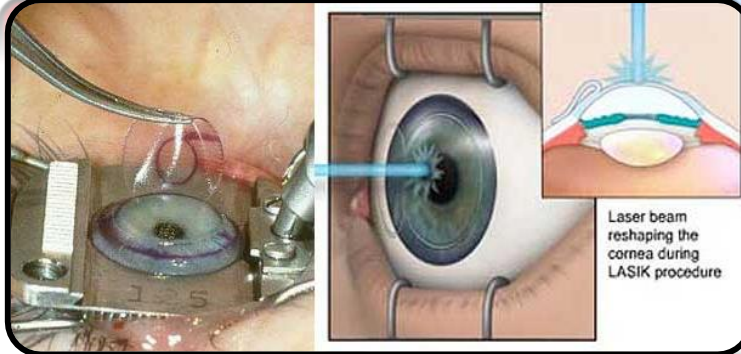
Flap Removed



LASIK

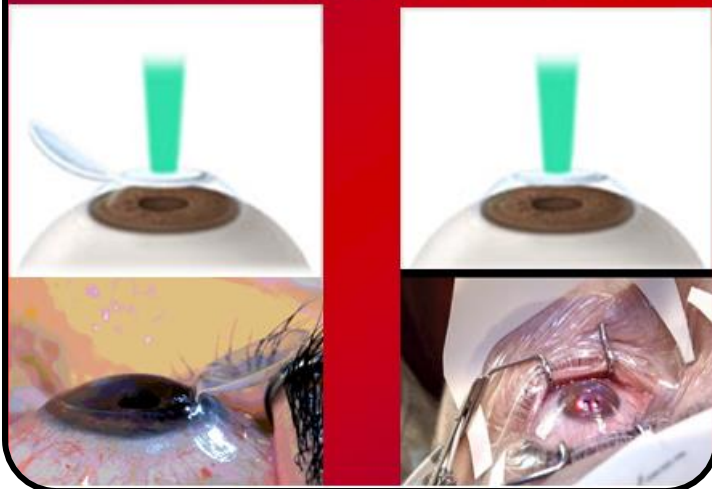
Flap Replaced

Post - Op.



Laser beam
reshaping the
cornea during
LASIK procedure

LASIK & Epi-LASIK (PRK)



LASIK & Epi-LASIK (PRK)



LASIK & Epi-LASIK (PRK)



LASIK & Epi-LASIK (PRK)



Therapeutic Uses

Anterior Segment

- 1) LASIK & PRK
- 2) YAG Capsulotomy

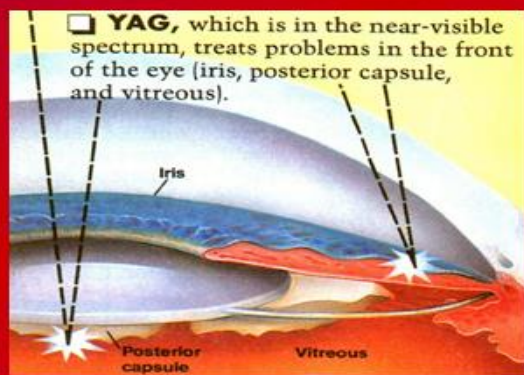
Therapeutic Uses

Posterior Capsule Opacity (After Cataract)



Therapeutic Uses

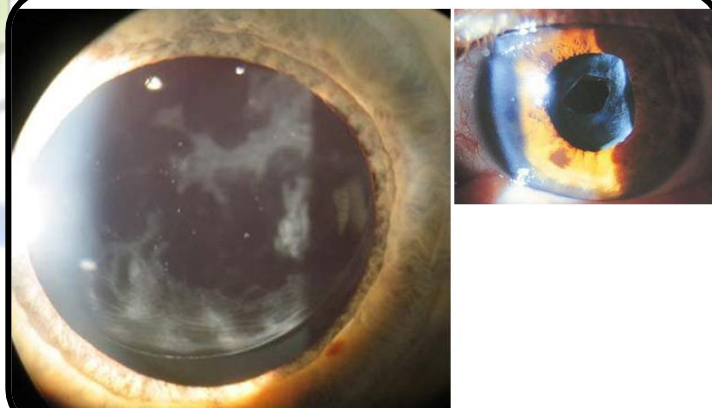
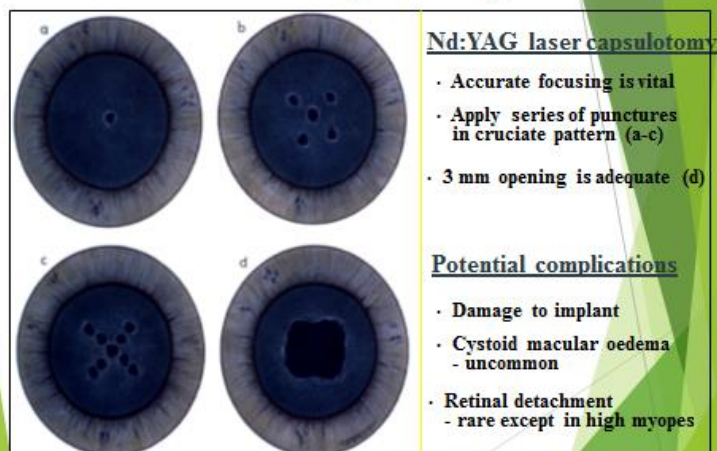
Posterior Capsule Opacity



YAG Capsulotomy



Treatment of capsular opacification



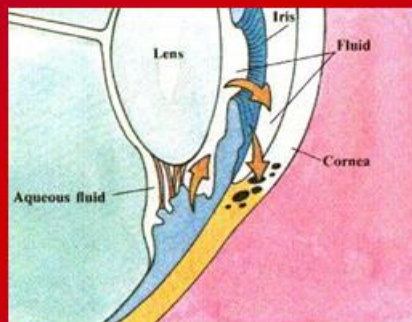
Therapeutic Uses

Anterior Segment

- 1) LASIK & PRK
- 2) YAG Capsulotomy
- 3) GLAUCOMA

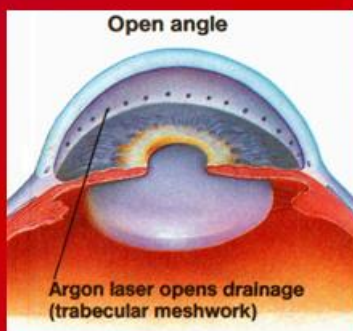
Therapeutic Uses

What is Glaucoma ?



Therapeutic Uses

Argon Laser Trabeculoplasty (ALT)



(trabecular meshwork)
Argon laser opens drainage

Therapeutic Uses

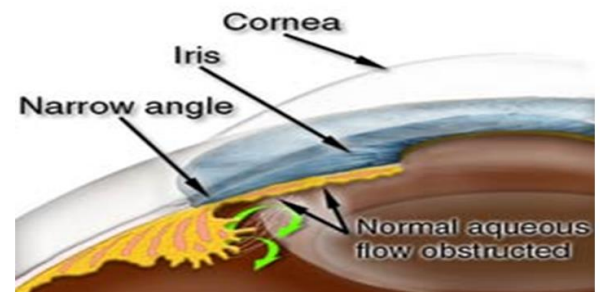
Argon Laser Trabeculoplasty (ALT)



Therapeutic Uses

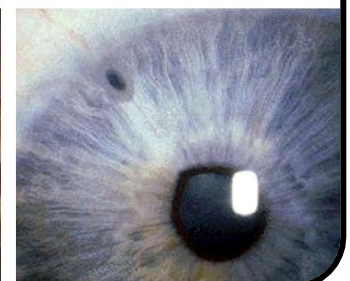
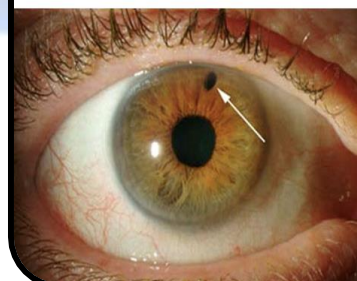
YAG Peripheral Iridectomy (PI)

Narrow Angle Glaucoma

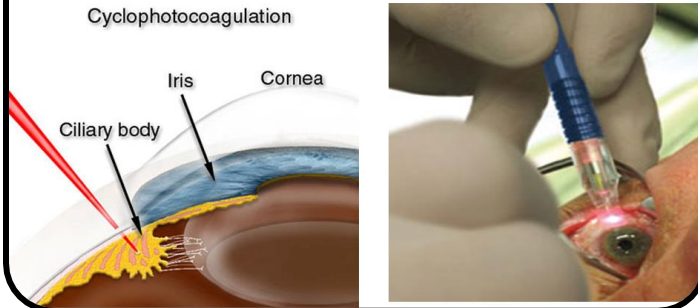


Therapeutic Uses

YAG Peripheral Iridectomy (PI)



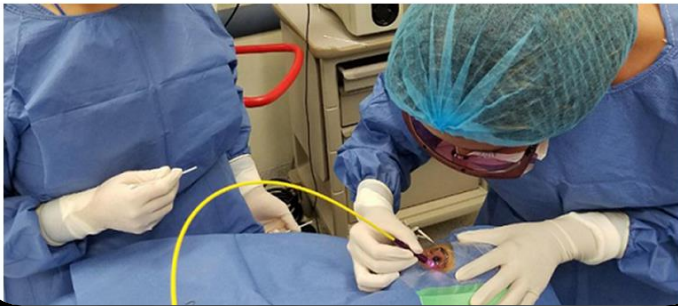
Therapeutic Uses Cyclophotocoagulation



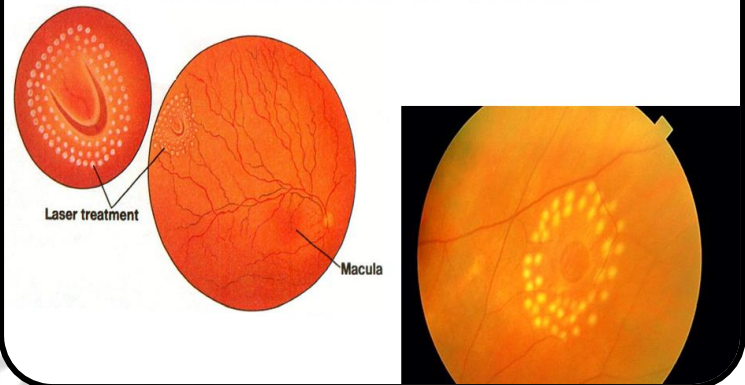
Therapeutic Uses Argon LASER



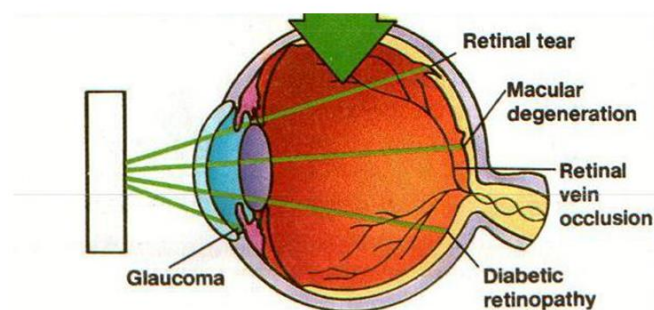
Therapeutic Uses Cyclophotocoagulation



Therapeutic Uses Retinal Breaks or Tears



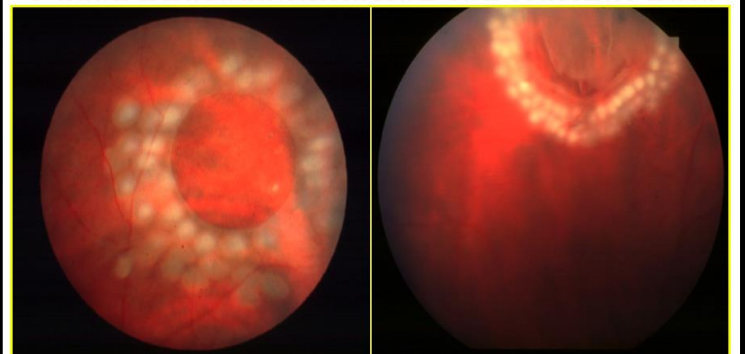
Therapeutic Uses Posterior Segment



Treats retinal tear, macular degeneration, diabetic retinopathy, glaucoma, retinal vein occlusion.

Therapeutic Uses

Laser photocoagulation for Retinal hole

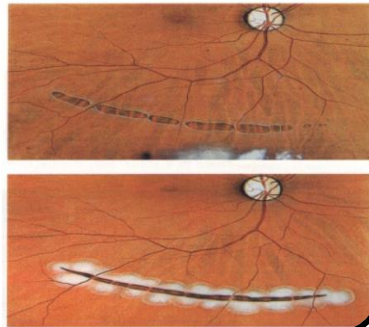
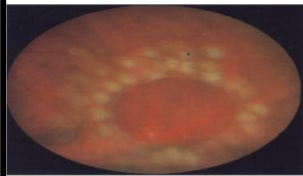


Surround lesion with two rows of confluent burns

Difficult for anterior lesions and if media hazy

Therapeutic Uses

Laser Retinopexy



Therapeutic Uses

What is Diabetic Retinopathy ?



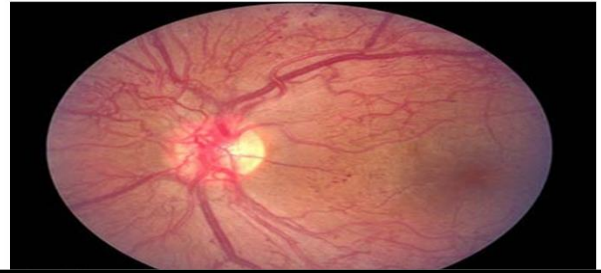
Therapeutic Uses

Diabetic Retinopathy



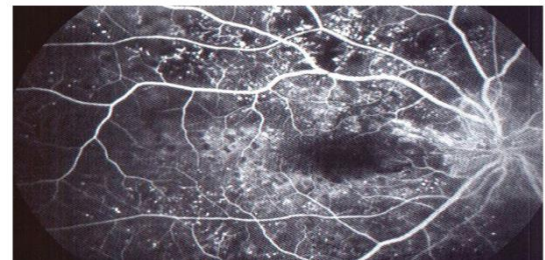
Therapeutic Uses

Diabetic Retinopathy



Therapeutic Uses

Diabetic Retinopathy



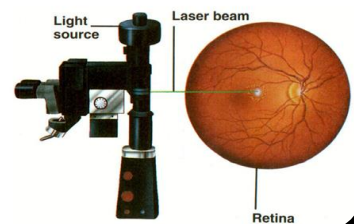
Therapeutic Uses

Diabetic Retinopathy

- Severity of DR runs parallel to the duration of DM
- 50 % of Diabetics have DR after 10 years of DM
- 90 % of Diabetics have DR after 20 years of DM
- A regular checkup every 6 - 12 months can detect early changes & prevent blindness

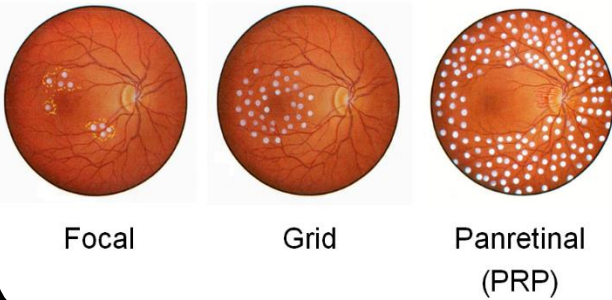
Therapeutic Uses

Diabetic Retinopathy



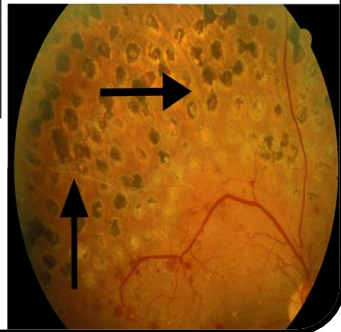
Therapeutic Uses

Diabetic Retinopathy

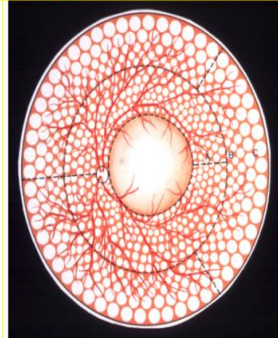
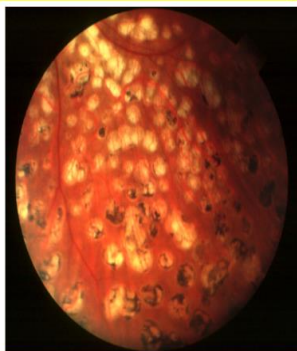


Focal

Grid

Panretinal
(PRP)Panretinal
Photocoagulation
(PRP)

Laser panretinal photocoagulation (PRP)



- Initial treatment is 2000-3000 burns
- Spot size (200-500 μm) depends on contact lens magnification
- Gentle intensity burn (0.10-0.05 sec)
- Area covered by complete PRP
- Follow-up 4 to 8 weeks

Contact intensity burn (0.10-0.05 sec)

on contact lens magnification

Spot size (200-500 μm) depends

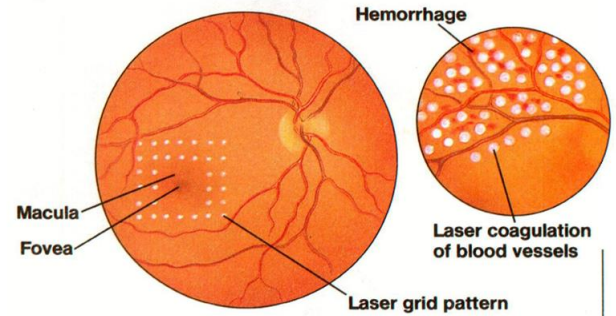
Initial treatment is 2000-3000 burns

Follow-up 4 to 8 weeks

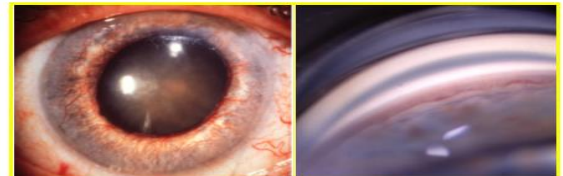
Area covered by complete PRP

Therapeutic Uses

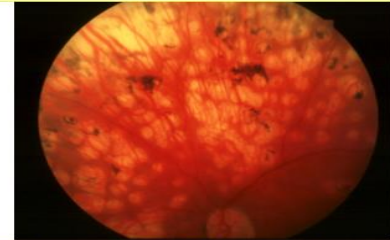
Retinal Haemorrhage



Management of Central Retinal Vein Occlusion



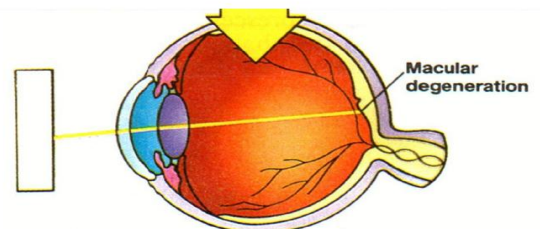
- Check every month for 6 months
- Look for rubeosis and angle new vessels



- Treat neovascularization by panretinal photocoagulation (PRP)

Therapeutic Uses

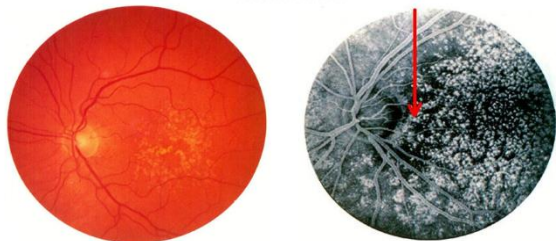
Photo Dynamic Therapy (PDT)



Treats macular degeneration.

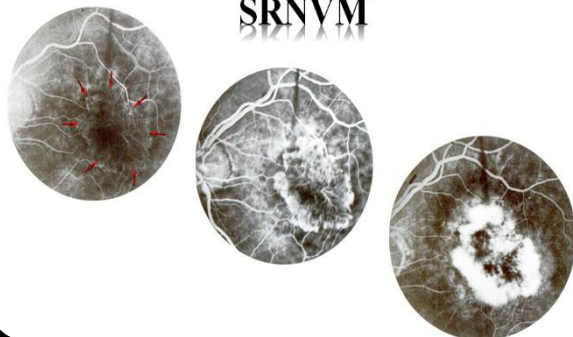
Age Related Macular Degeneration

Drusen



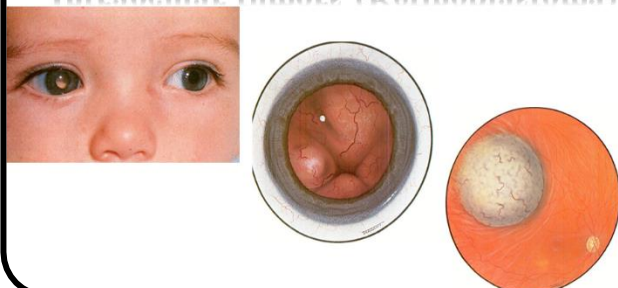
Age Related Macular Degeneration

SRNYM



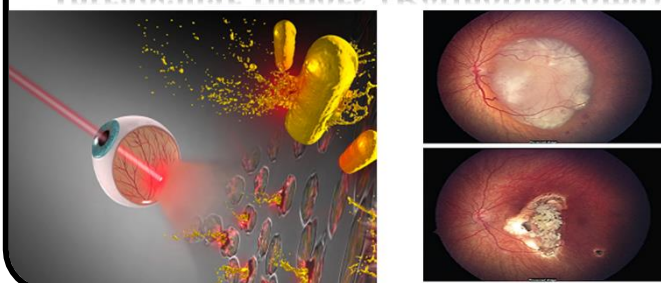
Therapeutic Uses

Intraocular tumors (Retinoblastoma)



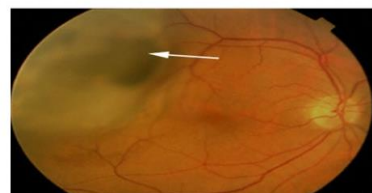
Therapeutic Uses

Intraocular tumors (Retinoblastoma)



Therapeutic Uses

Intraocular Tumors (Choroidal Malignant Melanoma)



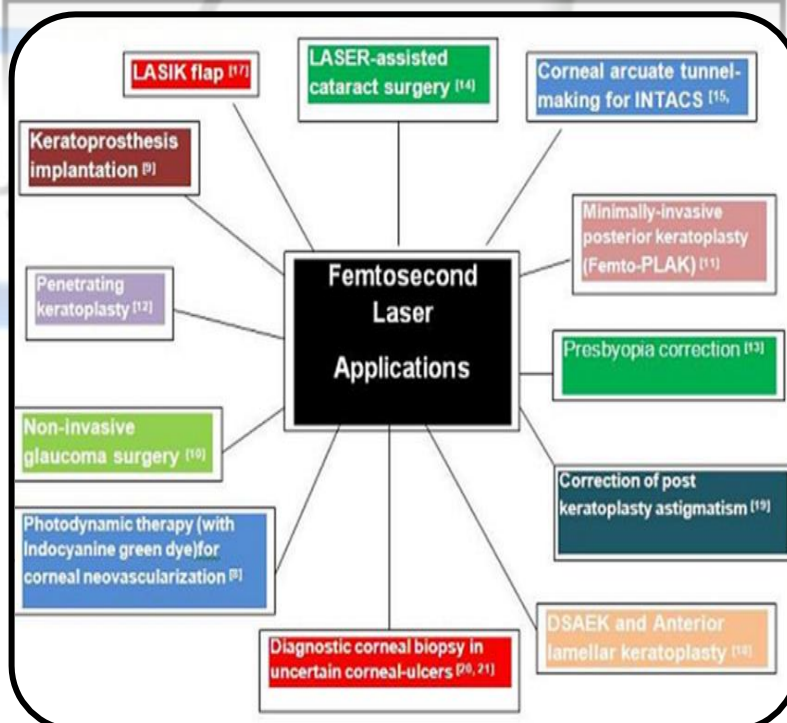
What is the Latest ?

- Femtosecond Laser
- Nanosecond Laser



What is Femtosecond Laser?

- Femtosecond lasers emit optical pulses of extremely short duration in the domain of femtoseconds, as short as one-quadrillionth of a second (10^{-15} sec).
- With these ultra-short pulses, tissue can be cut more precisely and with practically no heat development.
- The femtosecond laser is thus a highly accurate surgical laser which can be used in high precision ocular microsurgery.

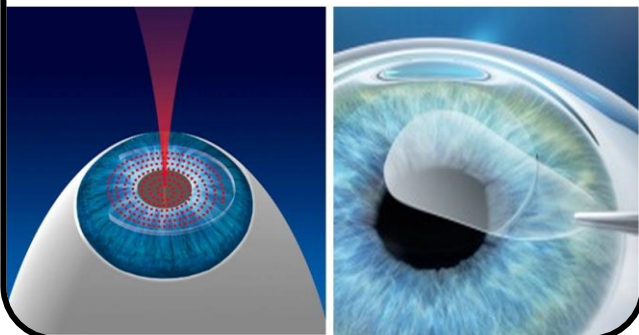




Femto LASIK



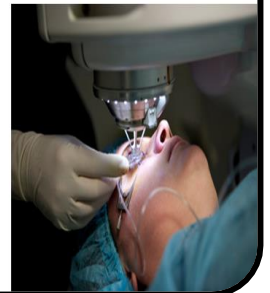
Femto LASIK & Femto Smile



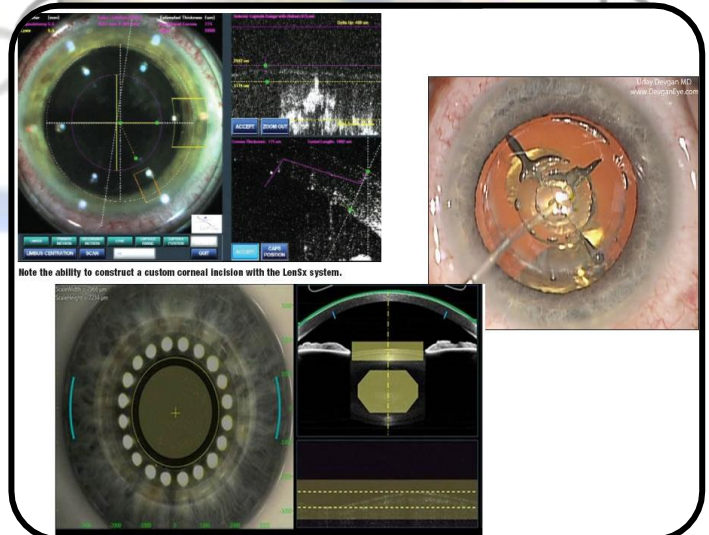
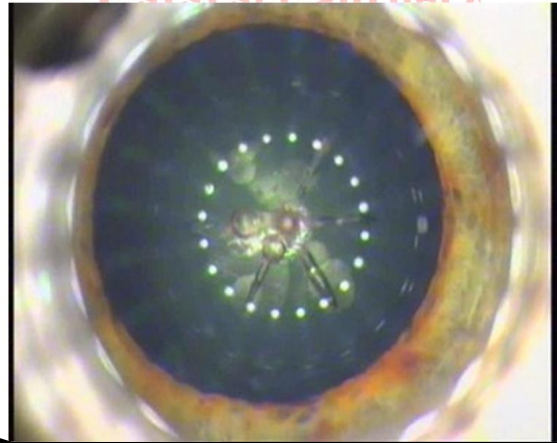
Femtosecond Cataract Surgery



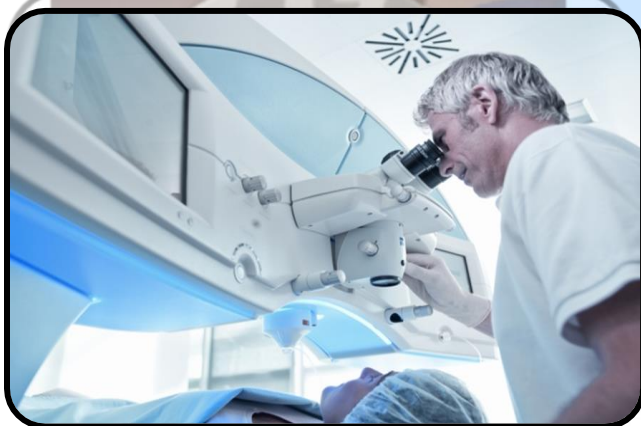
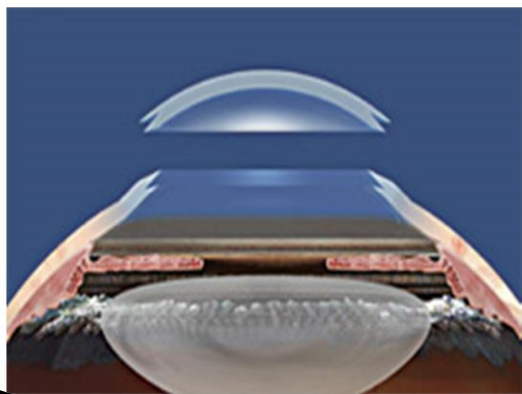
What Is Laser
Cataract Surgery?



Femtosecond Cataract Surgery



Femtosecond Corneal Graft Surgery



Lasers can....

- Save a child's eye as in Retinoblastoma
- Change a personality as in LASIK
- Cure a middle aged person with Glaucoma
- Restore Vision in a person with After-Cataract
- Preserve & Retain Vision in patients with DR & ARMD
- The possibilities are endless.....



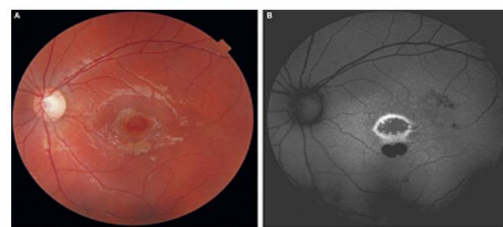
Complications Of LASER Photocoagulation

1. Wrongly photocoagulation of fovea, cornea, iris, or lens.
2. Choroidal effusion
3. Choroidal neovascularization
4. Peripheral visual defect
5. Dyschromatopsia

Lasers Pointer !!!



Lasers Pointer !!!



Refractive Errors

Dr. Horia Abdu Alla Alansi

The eye as an optical system:

Sensitive to the electromagnetic fields of light spectrum (visible light) in the range of 440 - 760 nm wavelength (λ)

Refractive property: Its refractive power (64 Diopters) is sufficient to bent rays and focus the image of an illuminated object on the sensitive retina (focal plan) which lie (axial length) at 22-24 mm from its focusing system (proportionate)

The focusing (refractive factors) are curvatures and refractive index (media density) focusing rays to the macula. 40% of D by the cornea and 18% by lens

The Normal State Of Refraction Or Emmetropia

In relaxed acc., the // rays are focused normally on the retina. The clear vision lies between far point at infinity and near point at 25cm in front of the eye for adults

Accommodation: lens function in which its power increases as we fixate to near objects by the synchysis of

- Increasing the anterior capsule convexity lens
- Miosis
- Convergence

This is mediated by the parasympathetic N. through 3rd nerve. It is more powerful in young ages.

Asthenopia: eye strain => headache, pain, lid margin redness, tearing and recurrent infections as sty.

Causes: 1- uncorrected Ametropia

2- Heterophoria (imbalance of AC/A=Acc-convergence Acc ratio) disappears by covering one eye

Refractive Errors

Incidence: 15 % needs glasses. Astigmatism with its myopic components are the major. Ethnicity as South East Asia >70%, civilization and reading are factor

With a relaxed accommodation; // rays from object, at infinity, are focused either in front (Myopia) or behind (Hypermetropia) the retina.

Emmetropia (E) is the normal refraction.

- D or Myopia M - if either (axial length or curvature) more than normal range.

+ D or Hypermetropia H - if either less than normal range.

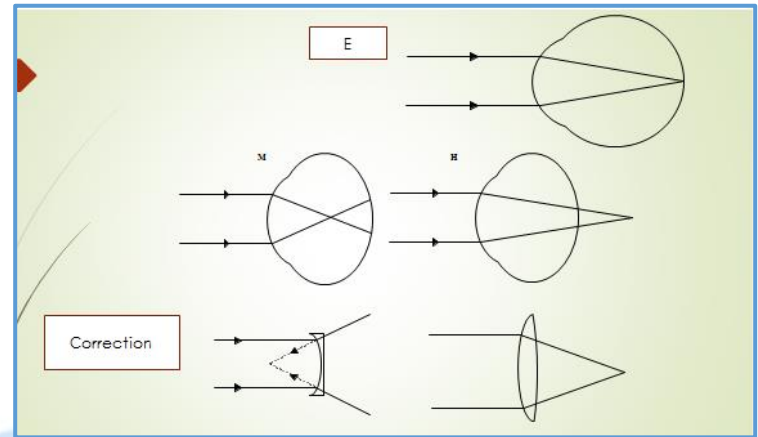
- **Axial types:** big eye => M, Small eye => H

- **Refractive types:**

- **Curvature:** Keratoconus => M, Cornea plana => H
- **Index:** refractive index, usually lens

Hypermetropia: posterior dislocation, hypoglycemia, aphakia

Myopia: anterior dislocation, hyperglycemia, early cataract



The Aim Of Correction:

is to put the image of an object at infinity (far point) at the fovea (Emmetropia) by choosing a lens (converging for H or diverging for M) with its focal points coincide with the eye far point.

e.g. an eye with far point 0.5m behind the eye (i.e H) is corrected by a spherical convex lens of +2.0D, with focal point at 0.5m as $D = 1/f$

Astigmatism Ast.: is due to difference in refraction between 2 meridians of the same eye. The commonest type of RE.

Regular Ast.: When the difference between the axes of least & greatest meridians is 90° with gradual transition in D-power. The correction need cylinder as its effective D power is only perpendicular to its axis (0 power // to the axis or no D).

Irregular Ast. If no perpendicular relation ship as in c. sarring o the cornea and ttt by hard contact lens if mild or keratoplasty if severe.

When vertical meridian power > horizontal this is called with - the-rule Ast. explained by eye lids moving always vertical, if the horizontal has more D > V = against the rule Ast.

Myopia: (Short Sightedness)

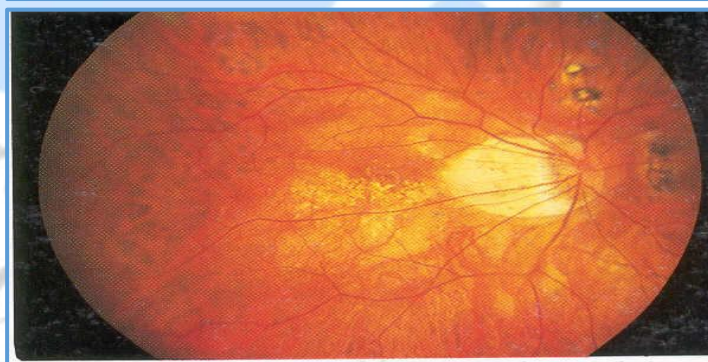
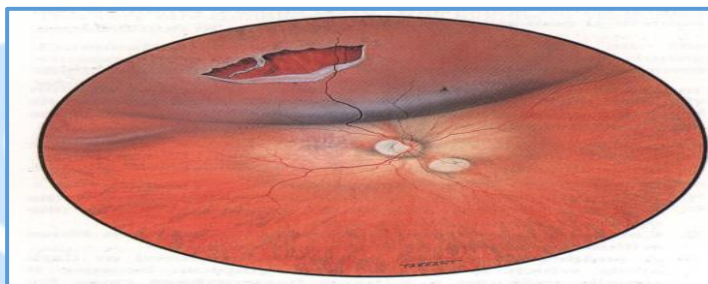
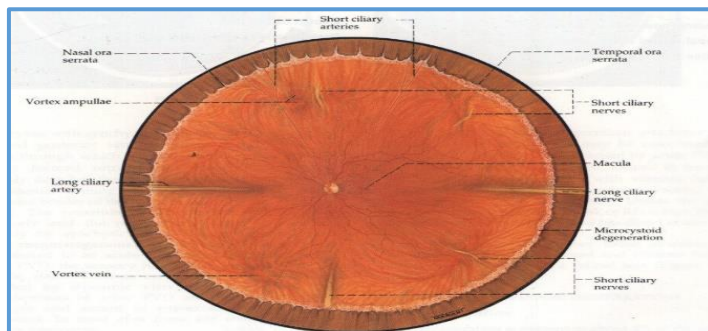
• Types:

- **Congenital M.:** rare, stationary up to - 10 D
- **Simple M:** commonest, onset at 14 years, to be stationary at 25, may reach - 6.0D
- **Axial M.** (High, progressive, degenerative, malignant, and pathological): Accompanied by organic changes as thin sclera, chorioretinal deg. syneresis...RD. up to -25 D.
- **Refractive: Curvature:** Keratoconus, lenticonus,

- **Index:** hyperglycemia, early cataract, drugs.
- **C/P:**
 - **Indistinct far vision**, the only symptom in simple M, improved by narrowing of the palpebral fissure,
 - **Asthenopia:** eyestrain: complains of headache, eye aches, lid margin redness, tearing and recurrent infections as sty.
- **DD:**
 1. Ametropia
 2. Heterophoria (imbalance of AC/A = Acc. convergence Acc. ratio),
 3. Presbyopia.

Axial M (Pathological):

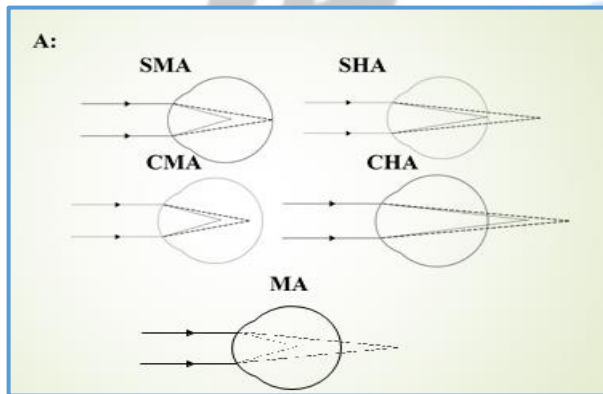
- progressive deteriorating vision, photophobia, musca volitans (vitreous deg-syneresis) and night blindness (peripheral retinal deg.)
- **Signs:** big eye, deep anterior chamber, apparent esotropia (negative angle alpha, or exophoria / exotropia (less Acc because clear near vision).
 - **Fundus exam:** chorioretinal deg., posterior staphyloma, 70% has peripheral lattice degeneration which may coalesce leading to retinal hole and retinal detachment (RD).
 - **Causes of blindness:** macular Hg & associated open angle glaucoma, RD, all result in optic atrophy.
 - **Diagnosis:** Retinoscopy: reveals against movements of the papillary red reflex neutralized by the appropriate concave lens.
- Autorefractometry:** - sphere > 12 D. with / without cylinder
- A- scan U/S => long A-p axis, B-scan => ~ Retinal Detachment.**
- **Ttt:** minus or concave glasses, contact lenses,
 - **Complication:** prophylactic retinopexy in lattice D. to prevent RD, retinal surgery for RD. intraocular pressure follow up.



Hypermetropia: (Long Sightedness)

- **Components;**
 - **latent H** corrected by the inherent ciliary muscle tone, **manifest H** according to the degree of H, may corrected partially or totally by Acc. => **facultative H**, its symptomatic part is called the **absolute H**.
 - **Latent H + manifest H = total H**
In children - at 2-5 years, the latent H, the ciliary muscle tone and Acc. are high so topical cycloplegic as atropine is necessary for diagnosis and correction of Am.
- **C/P:** Asthenopia in reading, defective far vision in high degree.
 - **Signs:** no signs in small degree, esotropia in children
 - **Axial H:** small globe, so infant refraction +3.0D is normal.
 - **Apparent exotropia** (+ angle alpha), esophoria (Acc use for near vision)

- **Retinoscopy:** with-shadow-movement throughout 360° of the eye pupil red reflex neutralized by the target convex lens.
- **Autorefractometry:** + D without cylinder, shallow anterior chamber & narrow angle by *gonioscopy*.
- **Fundoscopy;** tortuous blood vessels, shiny and picture like papillitis (pseudopapillitis).
- **Complications:** esotropia and amblyopia if not corrected in childhood and closed angle glaucoma in old age in axial H.
- **Ttt:** positive or convex glasses, contact lens, ttt esotropia early to prevent amblyopia with strict wear of glasses, prophylactic iridectomy to prevent acute glaucoma if shallow AC.



Astigmatism A.

- No point of focus due to different in position of focus of the horizontal and vertical meridians of the same eye. Regarding vertical lid movements the vertical assumed to be higher in D (with the rule Ast) if horizontal is found higher then the Ast called against the rule. The **regular type** maintain gradual transition between the least and greatest meridians. In **the irregular type**, the Am. axes are many & with no perpendicular relation-ship, e.g. advanced keratoconus, corneal scarring after corneal ulcer or corneal surgical repair...
- **C/P:** asthenopia in small degrees and blurred vision in high degrees.
 - **Refraction:** unequal power of refraction D &/or sign between 2 meridians
 - **Autorefractometry:** the need for cylinder + axis
 - **Placido' disc:** oval shaped corneal reflex rings normally circular rings
 - **Keratometry:** unequal corneal curvature D.
- **Complication:** as axial M or H according to the dominant Am.
- **Correction**

- ✓ **Glasses** Spherical convex (+ lens) or concave (- lens) for H or M respectively, when the Am is 360° equal e.g. +2.0D or - 2.0D
- ✓ **Cylinder (+, -)** for A. Its *power* is > to its *axis*, so its axis is adjusted to be at the emetropic meridian.
- ✓ **Contact Lens:** The number of CL chosen from a table compared to spectacle number, hard CL used for improving VA in keratoconus. S/E: intolerance, corneal abrasion and ulcer, allergy from preservatives and corneal edema from extended overwear because decreased atmospheric O2 perfusion to corneal tissue by CL.
- ✓ **Surgical:** The eye refraction should be stable for a 1.5 year.

✚ **Lasik:** a process of Laser assisted kerato meliosis keratotomy: adjusted remodeling of the corneal curvature to reach an Em. state. It is also valuable for elimination of the superficial corneal scar that causes irregular A. The cornea should have normal range in thickness, curvature either numbers and configurations.

✚ **ICL:** implantable chamber lens, a lens put in posterior chamber surgically over the crystalline lens = phakic IOL: as alternative to lasik or if high D, cylinder or measurements of cornea is not good for lasik. Depth of anterior chamber should be 3.5mm or more.

✚ **Intra-stromal ring:** in cornea to support its thin stroma in keratoconus.

✚ **Keratolpasty:** for deep, central corneal scars, advanced keratoconus and corneal dystrophies.

Anisometropia:

- The two eyes show different refraction powers.
- Anisikonia: difference in retinal image sizes. Depends on the degree and its onset, the vision could be:
 - **Binocular**, the difference is within the fusion limits of the brain
 - **Uniocular**, other eye is amblyopic (children=> strabismus)
 - **Alternating**, one eye is used at once.

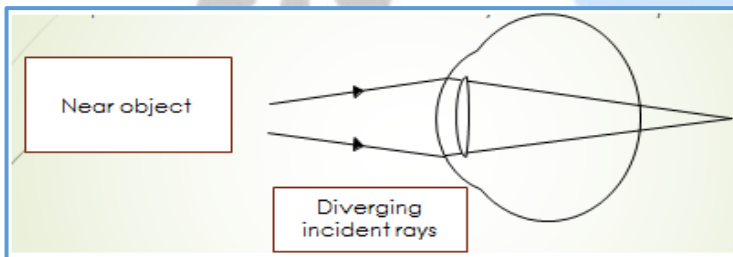
- **Diplopia**, if the difference is > 4.0 D. Usually in adults when there is no suppression mechanism, e.g removal of the lens.

Aphakia: an eye with no lens either congenital, surgical or traumatic dislocation.

The best correction is IOL (intraocular lens) or contact lens because retinal image magnification is 1%, 7% respectively that will not impair brain fusion of both eyes while +10.0 D glasses $\Rightarrow$ magnified image size by 25%, the brain cannot fuse this image with the other normal eye retinal image (diff image size = anisikonia). This leads to diplopia.

Presbyopia: blurring of vision when focusing near objects as reading due to physiological recession of the near point, usually at 40 years age in an Em person.

This follows the gradual decrease in Acc due to lens sclerosis & decrease of its elasticity e.g. at 60 years 1 D only available for Acc. (In childhood the near point was at 7 cm in front of the eyes +14 D Acc.)



Factors Affecting Age Of Presentation:

Type of RE: M will not complain of near vision difficulty if wearing ≥ 4 D glasses, lower power $\Rightarrow$ delayed presbyopia. In hypermetrope, earlier presbyopia than 40 years old.

Type of work: fine work at near will give complain earlier than mid distance worker.

C/P: uncomfortable reading (25-30 cm in front of the eyes, Acc $< +4.0$ D).

The letters seen unclear and running improved by strong light (miosis is a peripheral Acc stimulant) & keeping the book away by out stretching hands. It starts by asthenopia with reading at night.

Correction: reading-convex- glasses. Usually from 40 years old onward, we add a + 0.5D / 5 years age to the ordinary far glasses, so at 50 years +1.5D.

Keratoconus: due to keratoectasia $\Rightarrow$ progressive irregular conical shape of the cornea $\rightarrow$ progressive refractive changes in form of myopic astigmatism,

ppt factors: early ages allergic conjunctivitis and unstable refraction or the glasses change 2-3 x / yr getting higher number of cylinder D.

The latent form present in 13% of population and If diagnosed, it contraindicates Lasik correction otherwise normal refraction.

Signs: changing glasses number frequently /months or decreased vision progressively and increased myopic astigmatism correction, Mensen's sign (V-shaped lower lid when looking down), Kayser fissure ring from iron deposit around the cone from the tear film, scissoring refraction by retioscope and red reflex as oil droplet by ophthalmoscope.

Advanced stage: irregular Ast., vision is counting fingers not respond to glasses correction, Descement membrane rupture and painful hydropes that may end in scarring of cornea the treatment is keratoplasty.

Yong patient and suspected group needs serial refraction / 3months - 6months

Corneal curvature analysis by topography and pentacam
Corneal thickness by pachymetry normally > 530 mm

Treatment:

- **Optical**
- **Cross linking** by ultraviolet on a sensitized stroma by Vitamine B. for collagen bonds strengthening as soon as possible. Then follow up as the cross-linking may be repeated and this ttt strengthen collagens of cornea for life a
This is done under local anaesthesia to stabilize the corneal thickness and if attaining stable refraction, ICL inside the anterior chamber is the operation of choice if he wont to discard glasses.
- **Penetrating keratoplasty** for advanced KC. Rejection is not common as cornea is avascular.

Thank You

Vitreous Humour

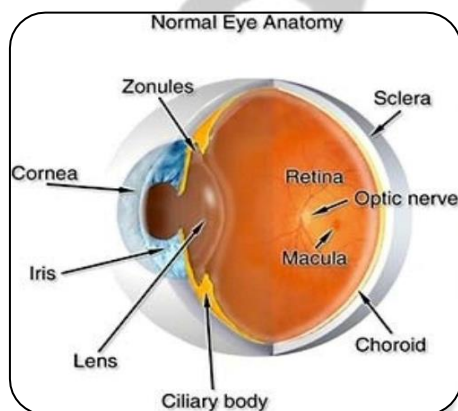
Dr .Ahmed M. Al-Asbahi

Ass.prof .Faculty of Medicine - Sanaa University

Vitreous Humour

Anatomy

- Vitreous body is transparent gelatinous substance that fills the space of the eye between lens and retina
volume 4ml(four-fifths of volume of eye ball)
- Weight 4,0g
- Refractive index 1,336



Structure Of The Vitreous

- Composed of a network of randomly - oriented collagen fibrils interspersed with numerous spheroidal macromolecules of hyaluronic acid
- The vitreous contains :** 99% of water, salts, sugars a network of collagen type 2 fibrils with glycosaminoglycon, hyaluronon and proteins

Parts Of Vitreous

Hyaloid Layer Or Membrane :

It is a true membrane Condensation of outer most surface layer of vitreous body .

It is divided into two parts:

1. Anterior hyaloid membrane

Starting from a point 1,5mm from ora serrata, is attached to the posterior lens

2. Posterior hyaloid membrane :-

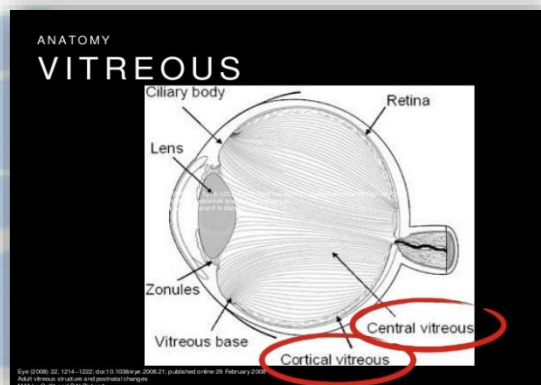
Extend back from the vitreous base up to optic disc, is loosely attached to the internal limiting membrane of the retina

Cortical Vitreous

- It is the outer part of the vitreous (hyaloid surface ,)Is 100 micron thick, Composed of densely packed collagen fibrils, cells, protein, and mucopoly saccharidy
- Metabolic center of the vitreous
- Subhyaloid space: between internal limiting membrane of retina and posterior hyaloid membrane.

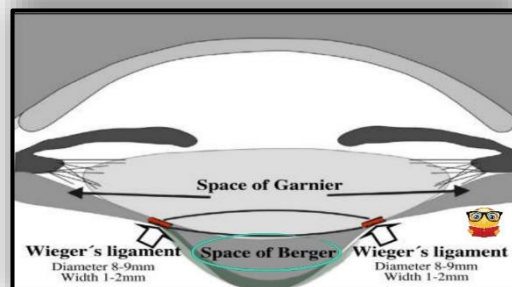
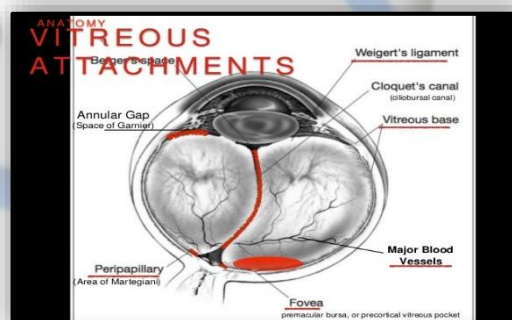
Medullary Vitreous(Nucleas)

Structurally it is similar to cortical vitreous except that it has a less fibrillar structure and does not contain hyalocytes.



Vitreous attachment:

- Vitreous base -strongest attachment,4mm wide,extending across ora serrata.
- Back of the crystalline lens (Ligament of Wieger)
- Around of the margin of optic disc-----Weiss ring
- Foveal region
- Retinal vessels



Function of vitreous:

- Maintains shape of globe and support retina in its normal position.
- Provide a clear media for optical transmission
- Screen out VU and IR light.
- Pathway for nutrients to reach the lens and retina.
- Diffusion barrier between the anterior and posterior segment of the eye.

Diseases Of The Vitreous

Persistent Hyperplastic Primary Vitreous PHPV

It is the most serious developmental disorder of the vitreous
It is caused by failure of regression of the primary vitreous:
can be divided into anterior and posterior PHPV

•Anterior PHPV :

C/P White pupil, microphthalmos, cataract secondary closed angle glaucoma.

Treatment: lensectomy and removal retrolental membrane

•Posterior PHPV :

C/P Retinal detachment and retinal dysplasia

Diagnosis considerations: clinical pictures, and Ultrasound

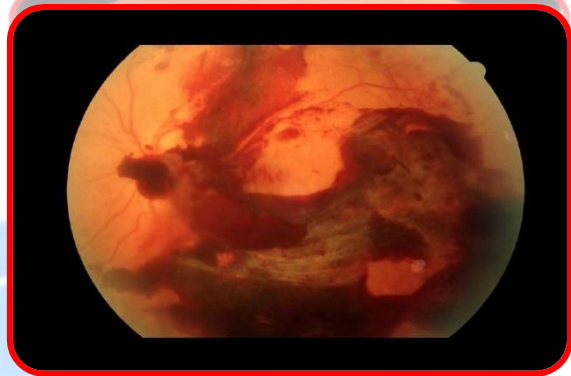
Treatment :treat RD

VITREOUS LIQUEFACTION

- Most common degenerative change in the vitreous
- Appearance of coarse aggregate material which moves freely in the free vitreous
- Associated with collapse (syneresis) and opacities in the vitreous ,black floaters in front of the eye

Vitreous Hemorrhage

- It is extravasation of blood into the vitreous body and its adjacent structures from retinal vessels, may present as pre-retinal Usually occurs (sub -hyaloid-)or intergel hemorrhage



Causes Of Vitreous Hemorrhage

Diabetic retinopathy

Retinal tear

Posterior vitreous detachment with vascular retinal tears

Rhegmatogenous retinal detachment

Proliferative sickle cell retinopathy

Macroaneurysm

Age related macular degeneration

Trauma

Clinical features

Sudden development of floaters--if small hemorrhage

Painless loss of vision --massive hemorrhage

Signs

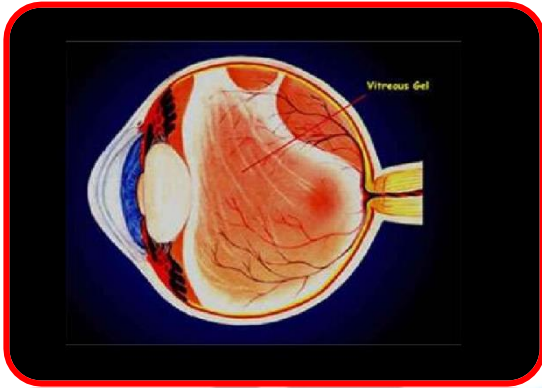
Distans direct ophthalmoscopy reveals black shadow against the red glow in small hemorrhage and no red glow in large hemorrhage
B Scan is particularly helpful

Treatment:

- Small vitreous hemorrhage may get absorbed but in recurrent hemorrhages retinitis proliferans may develop.
 1. Photocoagulation in cases of diabetic retinopathy.
 2. Vitrectomy

Posterior vitreous detachment

- Separation of the cortical vitreous from the retina
- **Symptoms:** Floaters or /and of light Sudden onset of flashes



- Blood in the vitreous
- Tumors calls: retinoblastoma
- Pigment- melenin

Symptoms

Muscae volitantes may appear in the form of black dots, lines or cobwebs.

When many vitreous opacities are present, there may be haziness of vision.

Signs:

- The opacities may be seen as black opacities
- independently of movements with distant direct of ophthalmoscopy

Treatment:

- Treatment of the underlying cause
- Vitrectomy

Posterior Vitreous Detachment

• Causes

1. Occurs in eyes with senile liquefaction, developing a hole in posterior hyaloid membrane .
2. The synchytic fluid collects between the posterior hyaloid membrane and the internal limiting membrane of the retina and lead to PVD
3. More common among aphakics and myopes
4. Trauma to eye

• PVD Complication

1. Retinal breaks
2. Vitreous hemorrhage
3. Retinal hemorrhage
4. Cystic maculopathy

VITREOUS OPACITIES

SYNCHYSIS SCINTILLANS

- Small white angular and crystalline bodies formed of cholesterol
- Trauma, vitreous hemorrhage or inflammatory disease
- Crystals sink in the bottom and stirred up with every movement
- "Beautiful shower of golden rain" on ophthalmoscopy
- Symptomless, untreatable

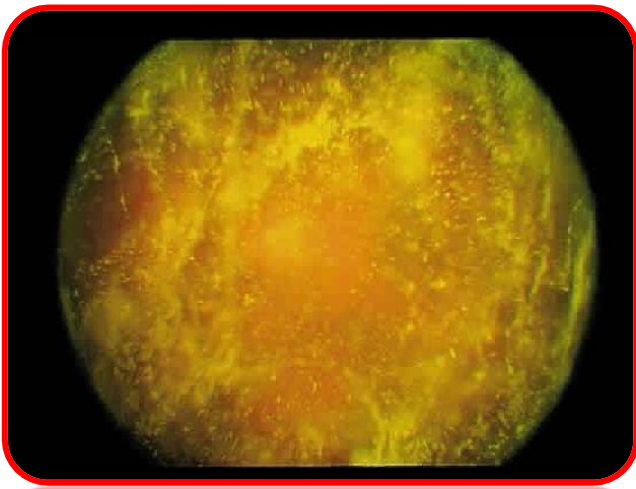
Vitreous Opacities

- Any opacity in the eye which casts a shadow upon the sentient elements of the retina (rods and cons) will appears as dark spot in the field of vision

• Causes:

1. Congenital:- remnants of hyaloid vascular system
2. Endogenous opacity:-
Coagula of the colloid basis of the gelatinous vitreous, degenerative myopia
Crystalline deposits –asteroid bodies, synchysis scintillans.
3. Exogenous opacities due to :-
protein coagula-plasmod vitreous as in iridocyclitis, and choroiditis





VITREOUS OPACITIES

ASTEROID HYALOSIS

- small, white rounded bodies suspended in the vitreous gel
- formed due to accumulation of calcium containing lipids
- unilateral, asymptomatic condition usually seen in old patients with healthy vitreous

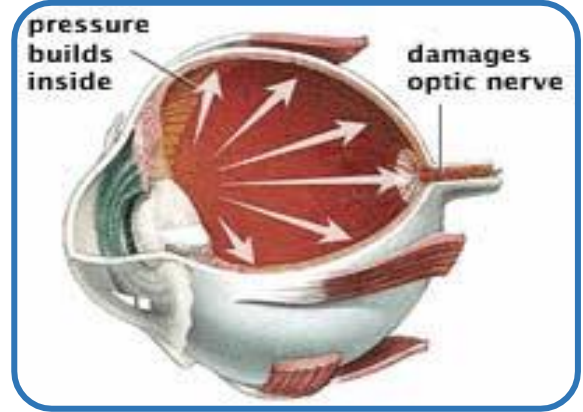
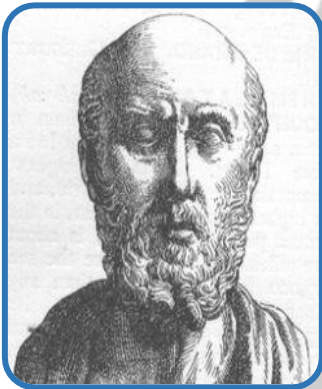


THANK YOU

Glaucoma

Historical Aspects

- The glaucoma term is derived from the old greek word "*glaukos*" which means "greyish-blue"
 - Hippocrates defined glaucoma as "a disease of the elderly patients in which the pupilla becomes blue".
- A person with a swollen cornea and a rapidly developing cataract and chronic (long-term) elevated pressure inside the eye



GLAUCOMA

What is it?

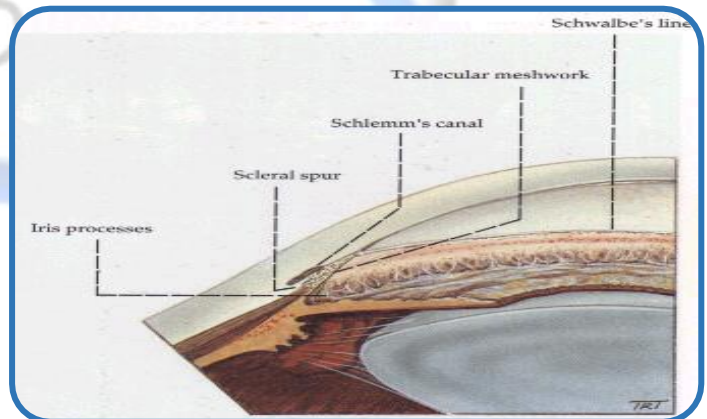
A disease of progressive optic neuropathy with loss of retinal neurons and their axons (nerve fiber layer) resulting in blindness if left untreated.

Glaucoma is a triad of:

1. Increase in IOP
2. Optic Disc Cupping
3. Visual Field Changes

Angle of the Anterior Chamber

- Situated between periphery of cornea and root of iris
- Drainage of the aqueous
- Structures:
 - The Trabecular Meshwork
 - Schlemm's Canal
- Examined by GONIOSCOPY



Introduction

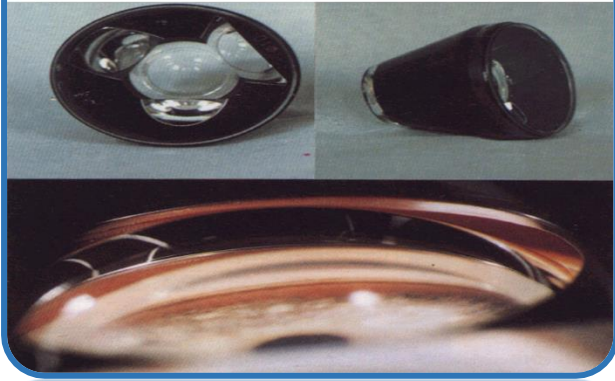
- According to WHO estimates, the most common causes of blindness around the developing world :
 1. Cataracts (47.9%)
 2. Glaucoma (12.3%)
 3. Age-related macular degeneration (8.7%)
 4. Corneal opacity (5.1%)
 5. Diabetic retinopathy (4.8%)
 6. Childhood blindness (3.9%)
 7. Trachoma (3.6%)

GLAUCOMA

"Glaucoma describes a group of diseases that kill retinal ganglion cells."

"High IOP is the strongest known risk factor for glaucoma but it is neither necessary nor sufficient to induce the neuropathy."

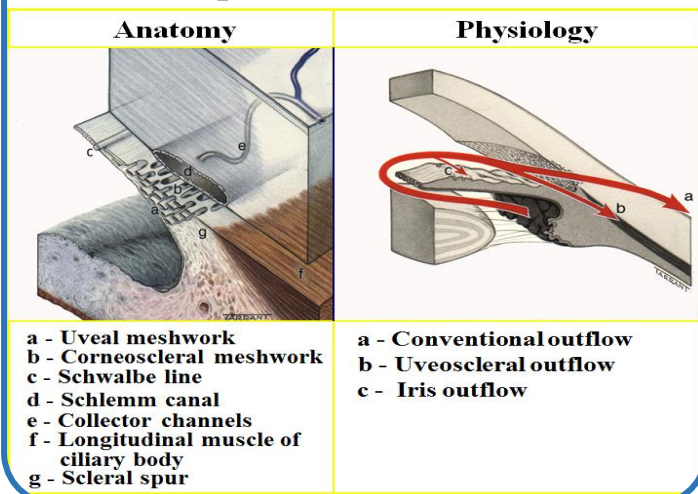
Goldmann triple mirror lens



Aqueous Humor

- Secreted by Ciliary Processes
- Drained through Trabecular meshwork
- AC Depth = 2.5 mm
- Secretion - Ciliary Processes
- Circulation
 - Posterior Chamber → Anterior Chamber
 - drained in the angle of Anterior Chamber
- Drainage
 - Pores of Trabecular meshwork → Canal of Schlemm → Aqueous veins → Episcleral Veins
 - Anterior Ciliary Veins.

Aqueous outflow

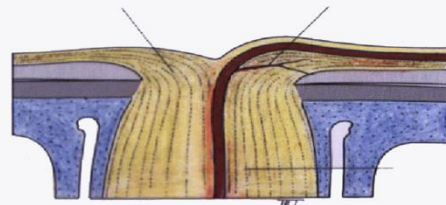


Glaucoma Pathogenesis

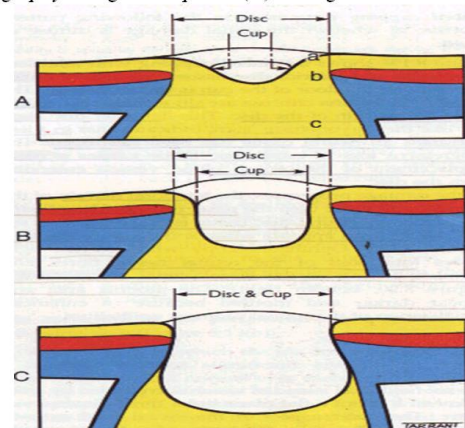
Pathogenesis of glaucomatous damage

- Direct mechanical theory
- Indirect ischemic theory
- the mechanism by which an elevated intraocular pressure damages nerve fibers:
 1. Raised IO pressure causes "mechanical" damage to the optic nerve axon
 2. Raised IO pressure causes "ischemia" of the nerve axon by reducing blood flow to it

Theories of glaucomatous damage



Cross section of optic nerve head
 (A) small physiological cup
 (B) large physiological cup and (C) total glaucomatous cupping



THE EVALUATION OF GLAUCOMA PATIENTS

1. Visual acuity (best corrected)
2. Biomicroscopy (clues to specific diagnosis...)
3. Measurement of intraocular pressure
4. Applanation tonometry (goldmann)
5. Noncontact tonometry
6. Pachymetry (central corneal thickness)
7. Evaluation of the anterior chamber angle (gonioscopy)

8. Visual field testing
9. Funduscopy

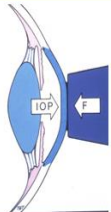
Glaucoma is a triad of:

- a. Increase in IOP
- b. Optic Disc Cupping
- c. Visual Field Changes

Measuring IOP

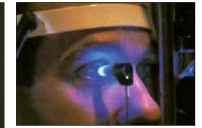
- Digital Method
- Tonometer
 - Indentation (Shiortz)
 - Applanation (Goldmann)
 - Air puff
 - Electronic

Tonometers

		
Goldmann Contact applanation	Perkins Portable contact applanation	Schiottz Contact indentation
		
Air-puff Non-contact indentation	Pulsair 2000 (Keeler) Portable non-contact applanation	Tono-Pen portable contact applanation



PERRKINS HAND
HELD TONOMETER



THE TECHNIQUES OF IOP MEASUREMENTS



NON CONTACT TONOMETER

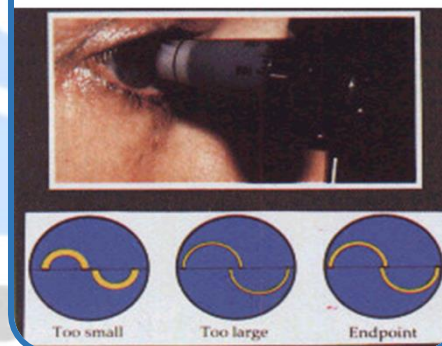


SCHIOTZ TONOMETER

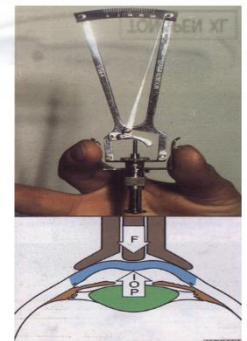


TONOPEN XL

Goldmann tonometer



Schiottz tonometer



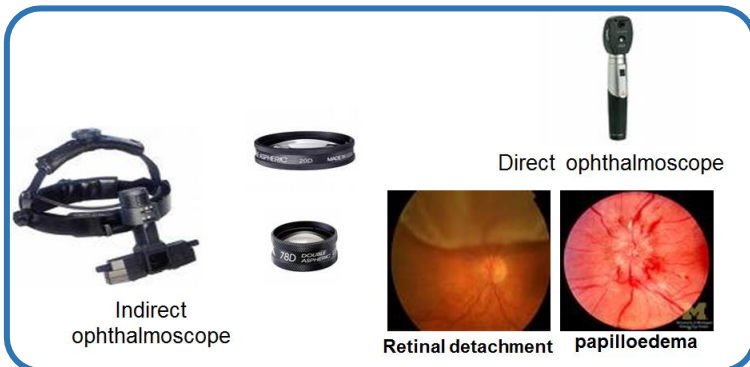
Glaucoma is a triad of:

1. Increase in IOP
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Funduscopy

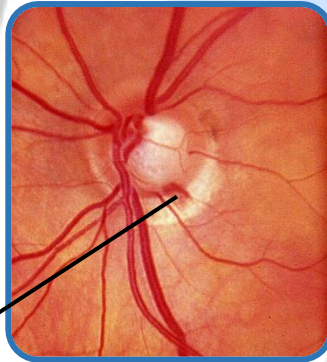
Retinal and optic disc exam need pupil dilatation

1. **Direct ophthalmoscope**
Unocular (no stereopsis) large image and small field and close to patient face
2. **Indirect ophthalmoscope**
Binocular (stereopsis= 3D) small image and large field need auxiliary lens (20D 28D)
3. **Slit lamp biomicroscopy**
Large image (fine retinal changes) with lenses (66D 78D 90)



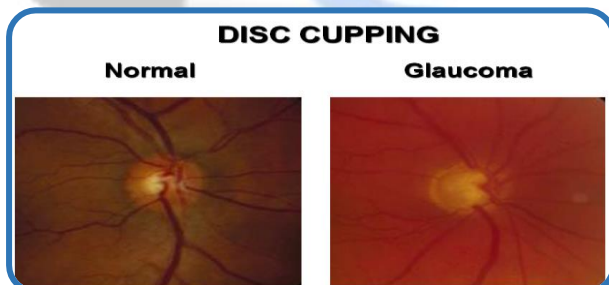
Five Rules for Assessment of the Optic Disc in Glaucoma

1. Observe the scleral Ring to identify the limits of the optic disc and its size
2. Identify the size of the Rim
3. Examine the Retinal nerve fiber layer
4. Examine the Region of parapapillary atrophy
5. Look for Retinal and optic disc hemorrhages

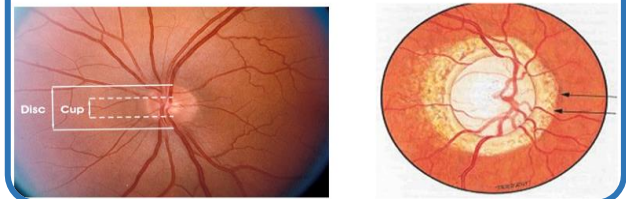


Optic nerve signs of glaucoma progression

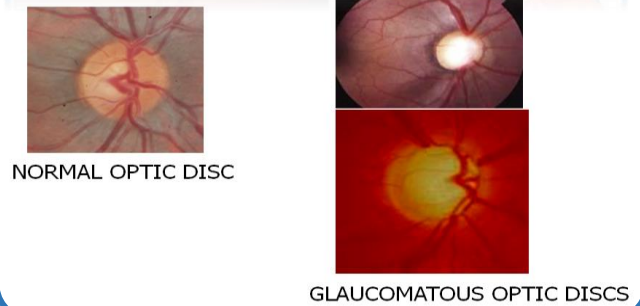
- ❖ Increasing C:D ratio
- ❖ Development of disk pallor
- ❖ Disc hemorrhage (60% will show progression of visual field damage)
- ❖ Vessel displacement
- ❖ Increased visibility of lamina cribosa



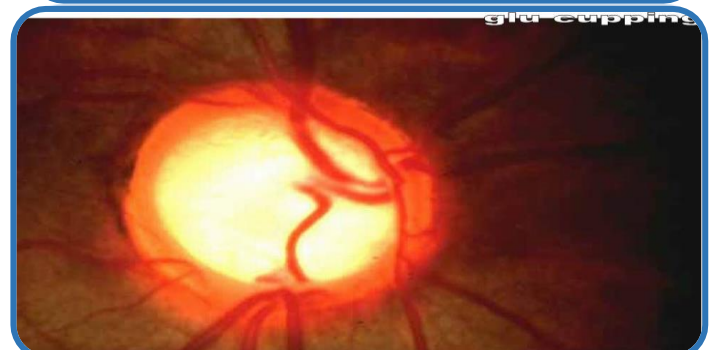
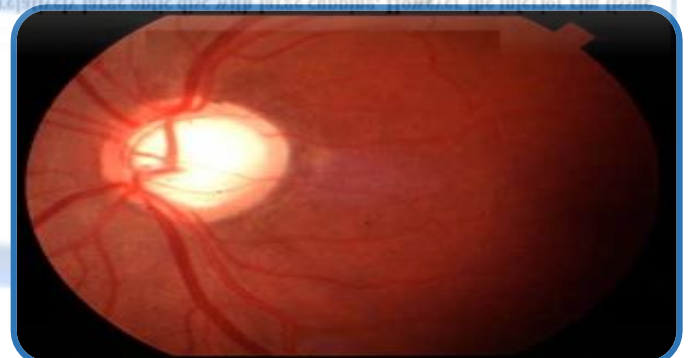
Cup-to-disk ratio

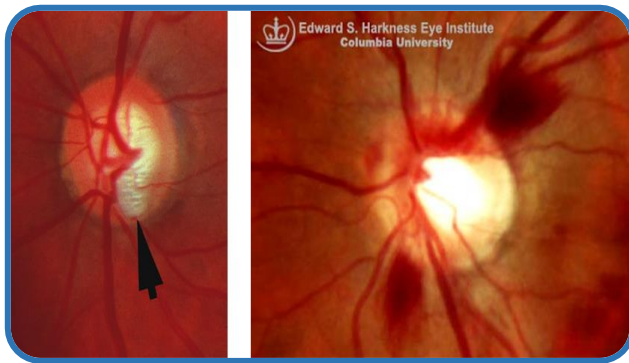


FUNDOSCOPIC CHANGES



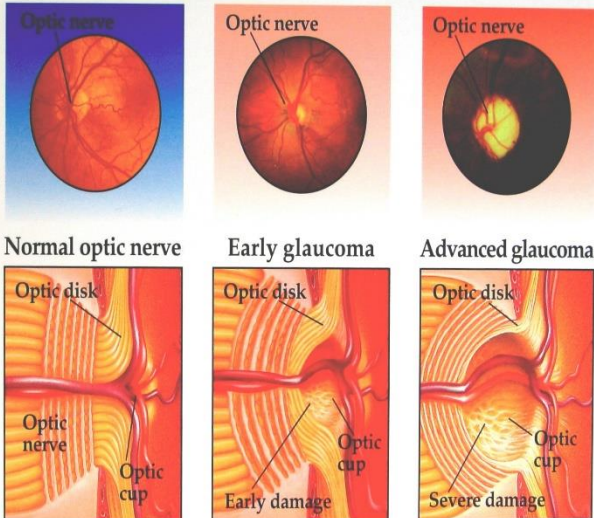
relatively large optic disc with large cupping. However the inferior rim tissue shows a localized notch





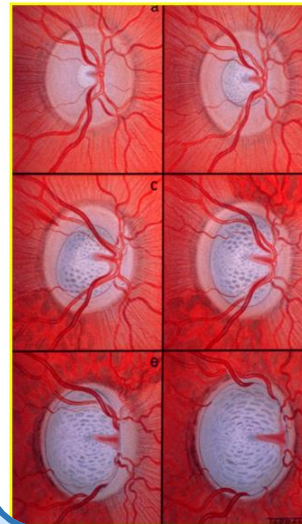
The Progression of Glaucoma

View of fundus



Glaucoma damages the optic nerve and leads to permanent loss of vision.

Progression of glaucomatous cupping



a. Normal (c:d ratio 0.2)

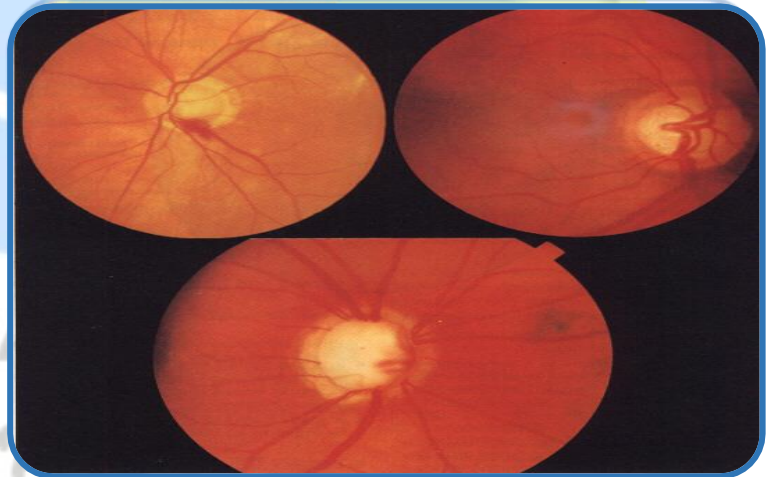
b. Concentric enlargement (c:d ratio 0.5)

c. AInferior expansion with retinal nerve fibre loss

d. Superior expansion with retinal nerve fibre loss

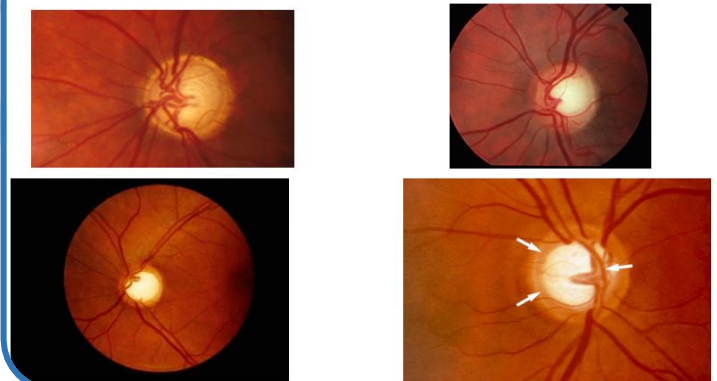
e. Advanced cupping with nasal displacement of vessels

f. Total cupping with loss of all retinal nerve fibres



GLAUCOMA

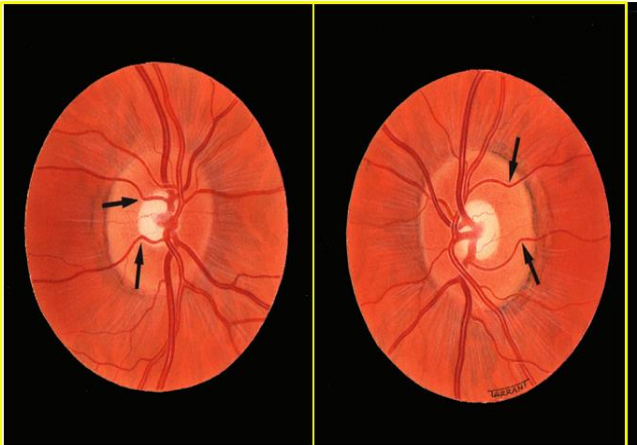
Glaucomatous cupping



Pallor and cupping

Pallor - maximal area of colour contrast

Cupping - bending of small blood vessels crossing disc



Cupping and pallor correspond. Cupping is greater than pallor.

Cupping and pallor correspond. Cupping is greater than pallor.

Glaucoma Is A Triad Of:

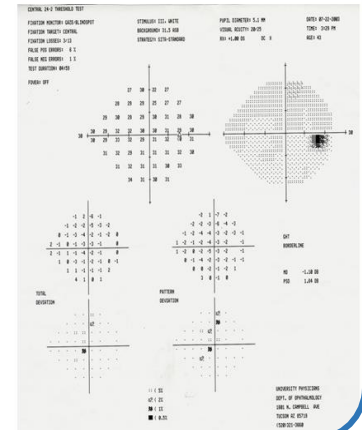
- Increase in IOP
- Optic Disc Cupping
- Visual Field Changes

Visual Field Testing

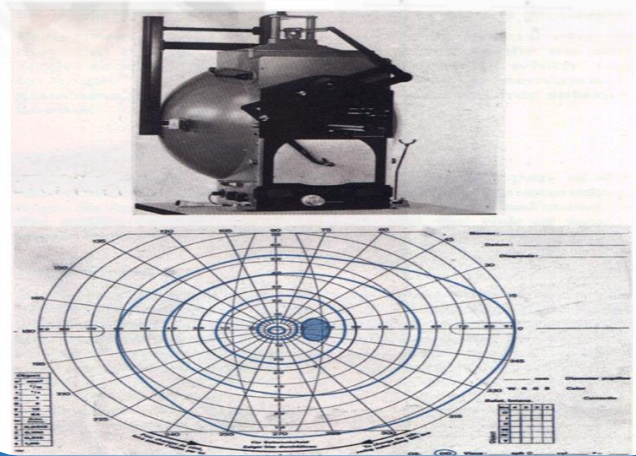
- VF testing to check the patients peripheral vision
- Typically by using an automated visual field machine
- This test is done to rule out any visual defects due to glaucoma
- VF defects may not be apparent until over 40% of the optic nerve fiber layer has been lost
- VF testing may need to be repeated
 - A low risk of glaucomatous damage, the test may be performed once a year
 - A high risk of glaucomatous damage, test may be performed as frequently as every 2 months

THE VISUAL FIELD

Humphrey automated perimetry

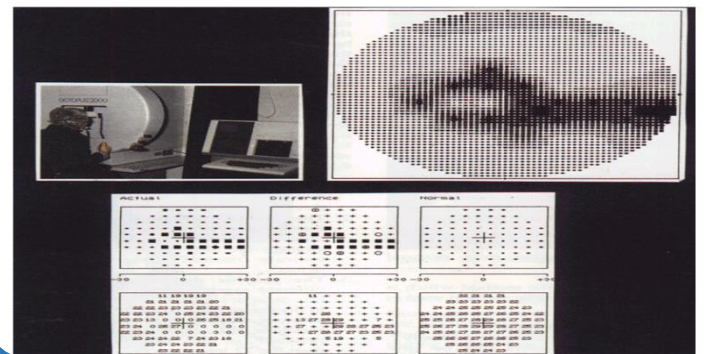


Goldmann perimeter



Automated perimeter

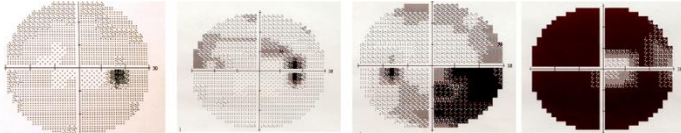
(graphic grey-scale, symbolic and numerical display)



DIFFERENT STAGES OF GLAUCOMATOUS VISUAL FIELD DEFECTS

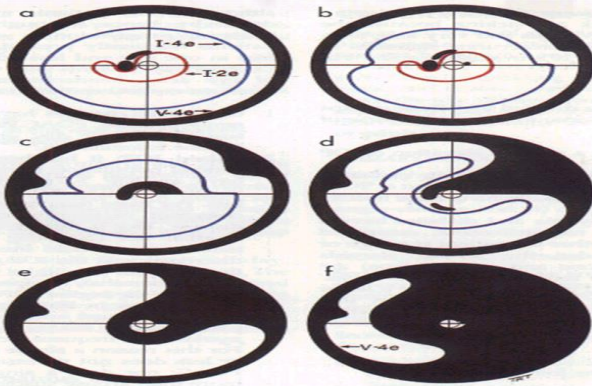


AUTOMATED VISUAL FIELD ANALYZER



NORMAL VF EARLY STAGE MODERATE STAGE END STAGE

Progression of glaucomatous visual field defects



Pachymetry

- Normal central corneal thickness is variable 500-520 microns
- Thinner cornea ($cct < 500 \mu m$) can give falsely low pressure readings
- Severe glaucoma patients may be failed diagnose
- A thick cornea ($>600 \mu m$) can give falsely high pressure readings
- Unnecessary treatments !!

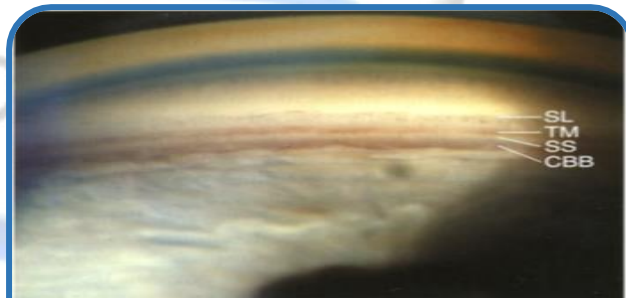
PACHYMETRY



Gonioscopy

Gonioscopy is performed to check the drainage angle of an eye

- A special contact lens (goniolens) is placed on the eye
- This test is important to determine if the angles are open, narrowed, or closed
 - Open angle: long term, slowly, insidious disease
 - Close (or narrowed): risk of acute glaucoma crisis



SL: SCHWALBE'S LINE
TM: TRABECULAR MESHWORK
SS: SCLERAL SPUR
CBB: CILIARY BODY BAND

CBB: CILIARY BODY BAND

SS: SCLERAL SPUR

TM: TRABECULAR MESHWORK

Classification of Glaucoma

- Congenital Glaucoma
- Acquired Glaucoma

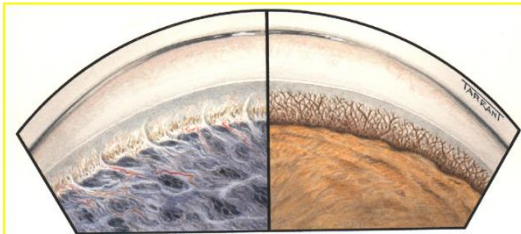
- Primary Glaucoma
 - Open Angle Glaucoma
 - Closed Angle Glaucoma
- Secondary Glaucoma

Congenital Glaucoma (Buphthalmos)

- Due to presence of an abnormal mesodermal membrane in the angle of AC
- 1:10,000 births
- More common in boys (65%)
- Symptoms & Signs
 - Early - Photophobia; hazy cornea
 - Late - Large eye (Ox Eye); Deep AC; Cupping discs

Primary congenital glaucoma

- Most sporadic - 10% autosomal recessive
- Absence of angle recess with iris inserted directly into trabeculum



Flat iris insertion

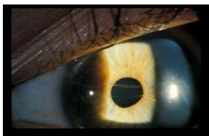
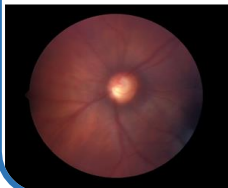
Concave iris insertion

Congenital Glaucoma

Buphthalmos, glaucomatous cupping, and cloudy cornea OD



Normal OS



Haab's striae



Clinical features of congenital glaucoma

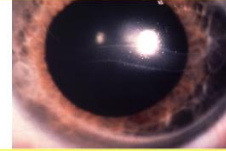
- Depend on age of onset
- Bilateral in 75% but frequently asymmetrical



Corneal oedema associated with lacrimation and photophobia



Buphthalmos if IOP becomes elevated prior to age 3 years.



Breaks in Descemet membrane



Optic disc cupping

Bilateral Buphthalmos (Haab's striae)

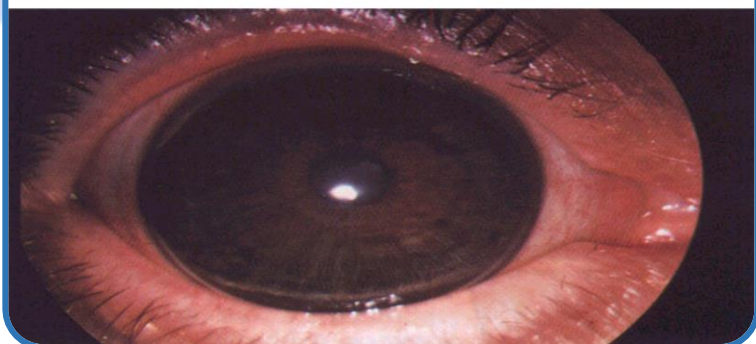


Congenital Glaucoma

Buphthalmos and cloudy corneas



Megalocornea



Acquired Glaucoma

CLASSIFICATION OF THE ACQUIRED GLAUCOMAS

PRIMARY OPEN ANGLE

GLAUCOMA

- NORMAL PRESSURE GLAUCOMA
- OCULAR HYPERTENSION

PRIMARY ANGLE CLOSURE

GLAUCOMAS

- ACUTE ANGLE CLOSURE GLAUCOMA
- SUBACUTE ANGLE CLOSURE GLAUCOMA

SECONDARY OPEN ANGLE

GLAUCOMAS

- PSEUDOEXFOLIATION GLAUCOMA
- PIGMENTARY GLAUCOMA
- PHACOLYTIC GLAUCOMA
- SECONDARY TO OCULAR INFLAMMATION
- SECONDARY TO HIGH EPISCLERAL VENOUS PRESSURE
- SECONDARY TO STEROID THERAPY

SECONDARY ANGLE CLOSURE

GLAUCOMAS

- DUE TO PERIPHERAL ANTERIOR SYNECHIAE
- SWOLLEN LENS OR PUPILLARY SECLUSION ANTERIOR MOVEMENT OF THE IRIS-LENS DIAPHRAGM
- **NEOVASCULAR GLAUCOMA**
- PLATEAU IRIS SYNDROME

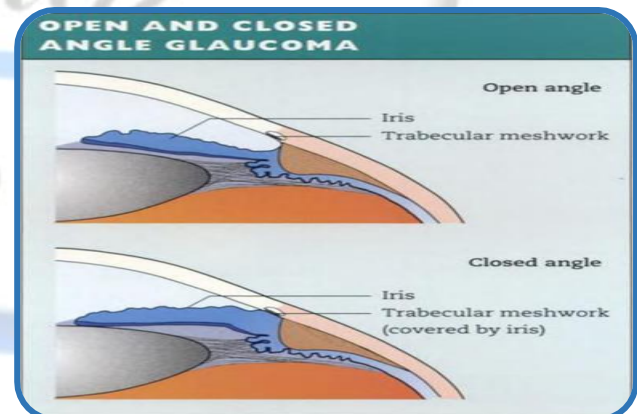
- SECONDARY TO STEROID THERAPY
- SECONDARY TO HIGH EPISCLERAL VENOUS PRESSURE
- SECONDARY TO OCULAR INFLAMMATION
- SECONDARY TO OCULAR

- PLATEAU IRIS SYNDROME
- **NEOVASCULAR GLAUCOMA**
- IRIS-LENS DIAPHRAGM
- SECLUSION ANTERIOR MOVEMENT OF THE

Primary Glaucoma:

This classification depends on:

- The iris doesn't cover the trabecular meshwork (*open angle*)
- The iris covers the trabecular meshwork (*closed angle*)



Open Angle Glaucoma

- Commonest type of glaucoma
- Affects 1 – 2 % of population above the age of 50 years.
- M=F
- Bilateral (one eye may be affected before)



Congenital Glaucoma

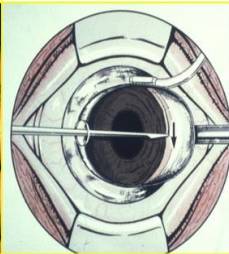
- Treatment
 - MEDICAL
 - SURGICAL (main treatment)

Management of Congenital Glaucoma

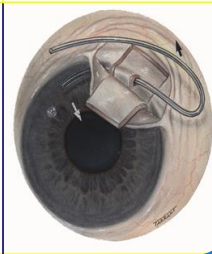
Measurement of IOP and corneal diameters



Goniotomy



Trabeculotomy



Open Angle Glaucoma (Chronic Simple Glaucoma)

- Slowly progressive elevation of IOP in absence of angle closure.
- Progressive cupping of optic disc and progressive deterioration of visual field.
- Due to obstruction of the outflow of aqueous (? sclerosis)

Primary open angle glaucoma

Pathogenesis:

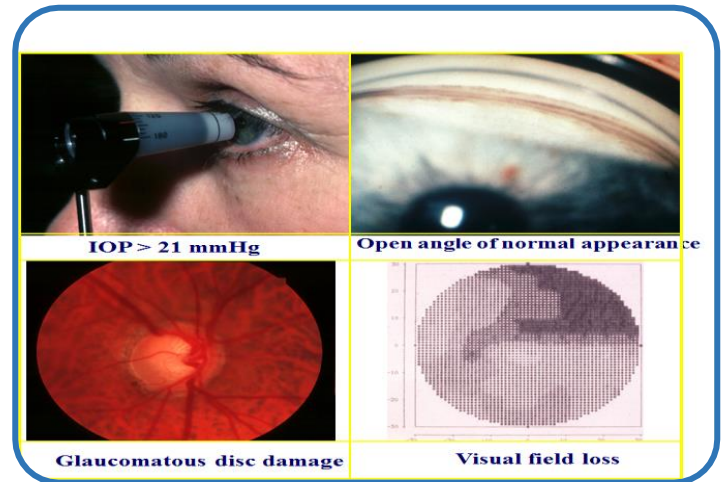
- The structure of trabecular meshwork appears normal but there is an increased resistance to the outflow, this happened due to:
 - Thickening of the trabecular lamellae which reduces the pore size
 - Reduction in the number of lining trabecular cells
 - Increased extracellular material in the meshwork spaces

Risk Factors

1. Age - most cases present after age 65 years
2. Race - more common, earlier onset and more severe in blacks
3. Inheritance
 - Level of IOP, outflow facility and disc size are inherited
 - Risk is increased by x2 if parent has POAG
 - Risk is increased x4 if sibling has POAG
4. Myopia

Open Angle Glaucoma

- Triad: IOP, Disc Cupping, Visual Field loss
- Symptoms:
 - Asymptomatic السويق
 - Some headache
 - Field loss
 - Night blindness
 - Complete visual loss in one eye

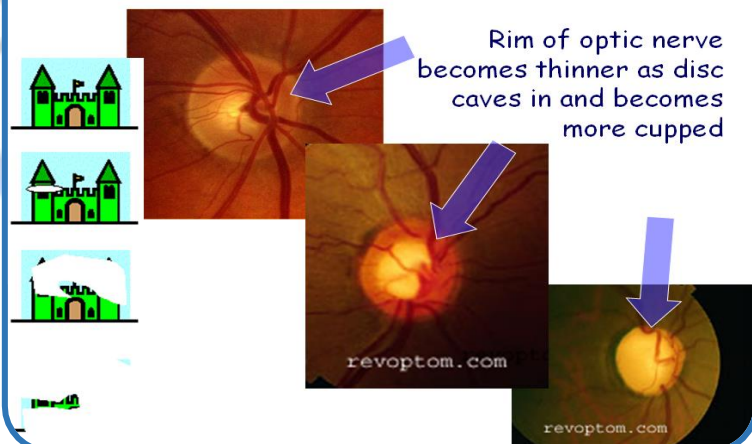


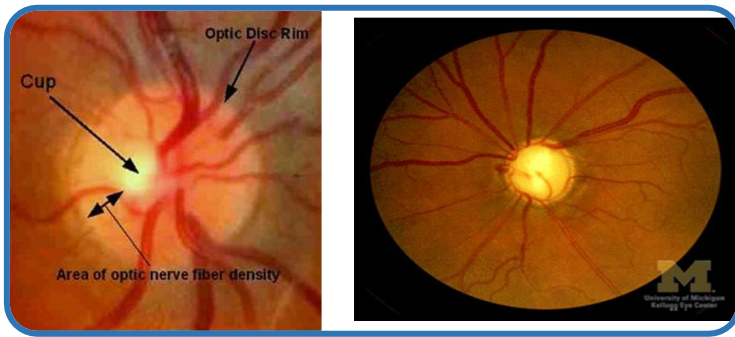
History:

- Symptoms depends on the rate of IO pressure rises.
- Associated with slow rise in pressure and it's symptomless until pt becomes aware of visual deficit.
- Many pts diagnosed via an optometrist.



Characteristic pattern to loss of visual field





Treatment of Open Angle Glaucoma

- Medical
 - Miotics (Pilocarpine)
 - Beta Blockers (Timolol, Betaxolol)
 - Prostaglandin analogues (Xalatan, Traphatan)
 - Acetazolamide (oral, drops)
- Surgical
 - Trabeculectomy
 - Trabeculectomy + Antimetabolites
 - Argon Laser Trabeculoplasty

FORMULA FOR TARGET IOP

- Rule of thumb
 - 20% reduction for mild cases.
 - 30 % for moderate cases.
 - 40 % for severe cases.

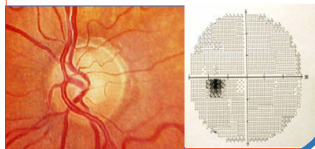
TWO DIFFERENT SITUATION

NORMAL TENSION (OR LOW TENSION) GLAUCOMA

- PEOPLE CAN HAVE OPTIC NERVE DAMAGE WITHOUT HAVING ELEVATED IOP
- THE MAIN REASON OF THIS CONDITION IS VASCULAR INSUFFICIENCY (OCULAR ISCHEMIA?)

OCULAR HYPERTENSION

- PEOPLE CAN HAVE ELEVATED PRESSURES WITHOUT SIGNS OF OPTIC NERVE DAMAGE OR VISUAL FIELD LOSS
- THEY ARE CONSIDERED AS A RISK FOR GLAUCOMA
- THESE PEOPLE ARE KNOWN AS GLAUCOMA SUSPECT



Normal Tension Glaucoma (NPG, LTG, LPB, NTG)

- Similar to OAG but IOP always < 21 mmHg
- Higher prevalence of vasospastic disorders, blood dyscrasias, autoimmune diseases
- May be related to episodic hypotension, hypothyroidism
- A diagnosis of exclusion!!!

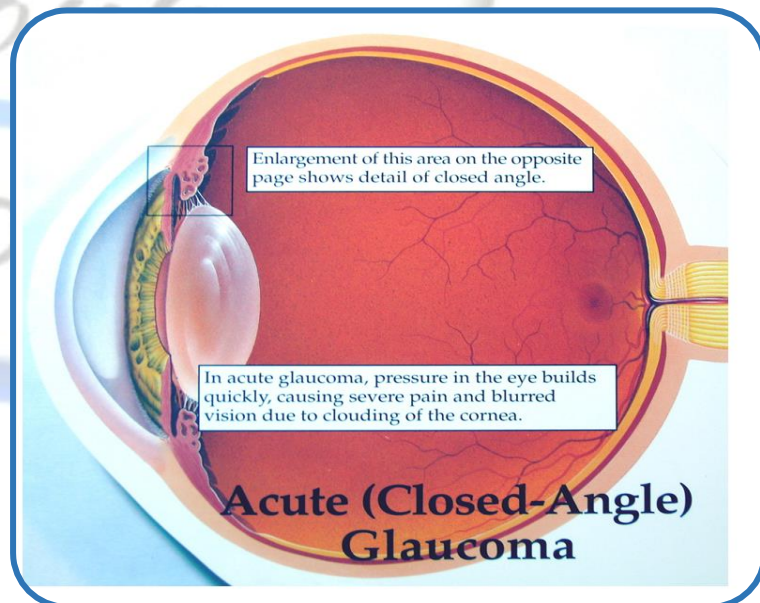
Low Tension Glaucoma

- IOP normal but cupping of disc and field loss
- Ocular Hypertension
 - IOP is high (>21 mm Hg) but disc and field are normal

Normal/low tension glaucoma

- when the optic nerve head is susceptible to IO pressure, damage happens even when the IO is NL
- This type of glaucoma is difficult to treat, although lowering the IOP may be beneficial !!
- **Ocular Htn:** IO pressure is raised but no optic disc damage

Closed Angle Glaucoma



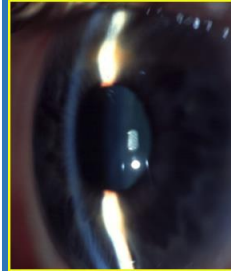
Closed Angle Glaucoma

- Results from closure of an already narrow anterior chamber angle
- Bilateral (affects one eye before the other)
- Above the age of 40
- Females > Males
- More in Hypermetropia
- Occurs in small eyes (hypermetropic) → shallow anterior chamber
- In response to pupil dilation the iris is bunched → pressure increased → bowing of the iris forward and closing the drainage angle... peripheral iris contact ultimately leads to adhesion (peripheral anterior synechiae).
- Aqueous circulation is reduced → no nutrition to the cornea and no O₂ delivery to the posterior cornea → corneal edema → more rise of IO pressure → reduced vision .
- Associated with transient rise of pressure...headache...colored haloes around bright lights during attacks

Classification

1. Latent - asymptomatic
 - IOP may remain normal
 - May progress to subacute, acute or chronic angle closure
2. Subacute - intermittent angle closure
 - May develop acute or chronic angle closure
3. Acute
 - Congestive - sudden total angle closure
 - Postcongestive - follows acute attack
4. Chronic - 'creeping or latent' angle closure
 - Follows intermittent angle closure
5. Absolute
 - No PL following acute attack

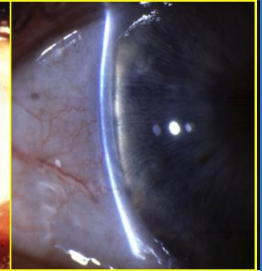
Anatomical predispositions



• Convex iris-lens diaphragm



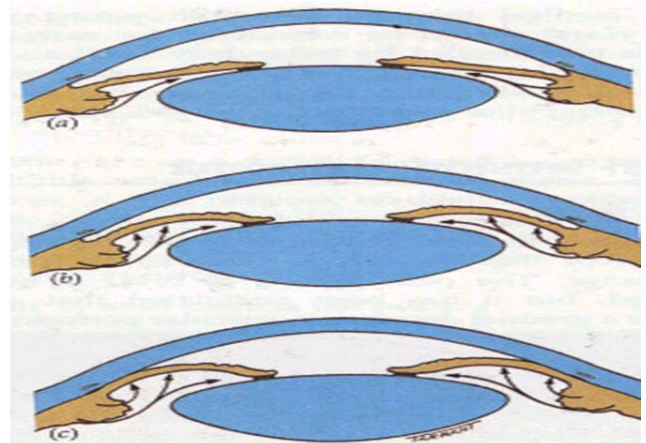
• Shallow anterior chamber



• Narrow entrance to chamber angle

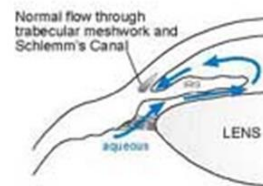
Mechanism of angle closure

(a) relative pupil block; (b) iris bombé; and (c) iridotrabecular contact

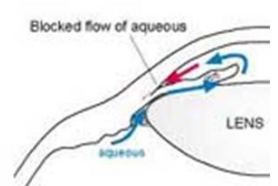


Closed angle glaucoma

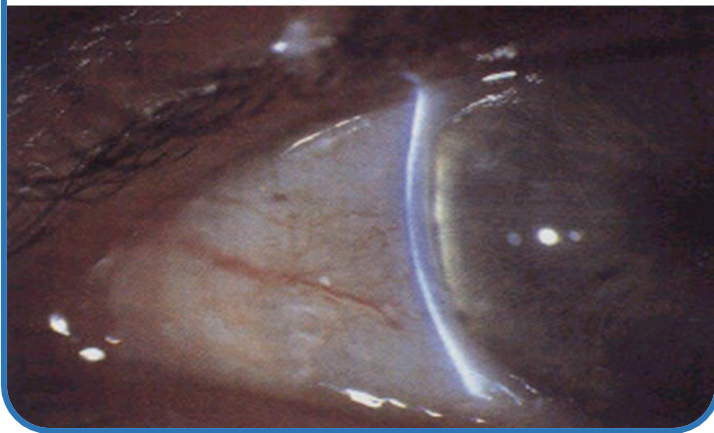
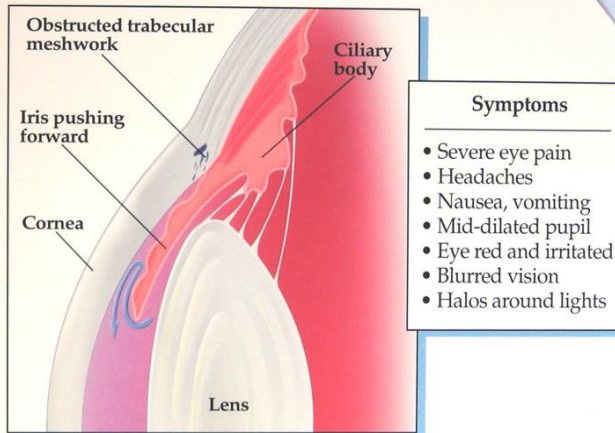
Angle Closure Glaucoma



Normal flow through trabecular meshwork and Schlemm's Canal



Blocked flow of aqueous

Close proximity of iris to peripheral cornea**Symptoms of Closed-Angle Glaucoma**

The angle between the iris and the cornea narrows or closes, obstructing the drainage of aqueous humor.

Clinical Picture

- Prodromal Stage
 - Transient attacks of elevated IOP
 - Headache
 - Blurring of vision
 - Colored haloes
- Acute Stage (Acute Congestive Glaucoma)
 - Pain
 - Vomiting
 - Decreased vision
 - Lid oedema, corneal oedema, AC shallow, mid-dilated pupil, stony hard, hazy view of fundus

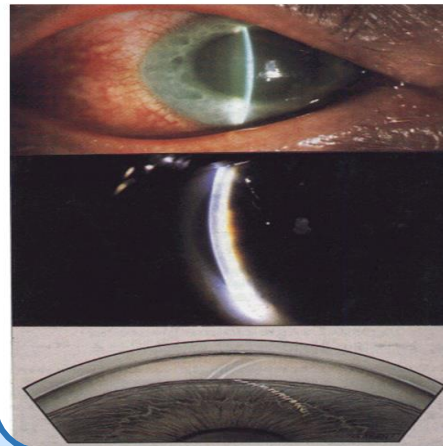
GLAUCOMA

Optic nerve signs of glaucoma progression

- ❖ Increasing C:D ratio
- ❖ Development of disk pallor
- ❖ Disc hemorrhage (60% will show progression of visual field damage)
- ❖ Vessel displacement
- ❖ Increased visibility of lamina cribosa

Clinical Picture

- Chronic Stage
 - Peripheral anterior Synechia
 - Glaucomatous cupping and
 - Visual field loss
- Absolute Stage
 - Eye is blind and painful

Acute congestive angle-closure glaucoma

Lid Oedema
Ciliary Congestion
Corneal Oedema
AC shallow
PAS
Pupil mid-dilated
Lens – Cataract
IOP – Stony hard
Fundus – cupping

Narrow Angle Glaucoma

Onset: 50+ years of age

Symptoms

Severe eye/headache pain
Blurred vision
Red eye
Nausea and vomiting
Halos around lights
Intermittent eye ache at night

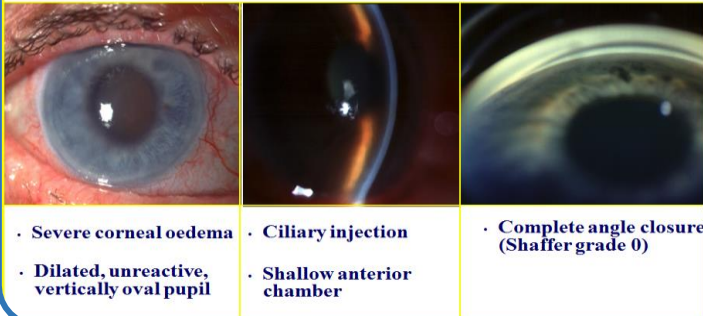
Signs

Red, teary eye
Corneal edema
Closed angle
Shallow AC
Mid-dilated, fixed pupil
“Glaucomflecken”
Iris atrophy
AC inflammation

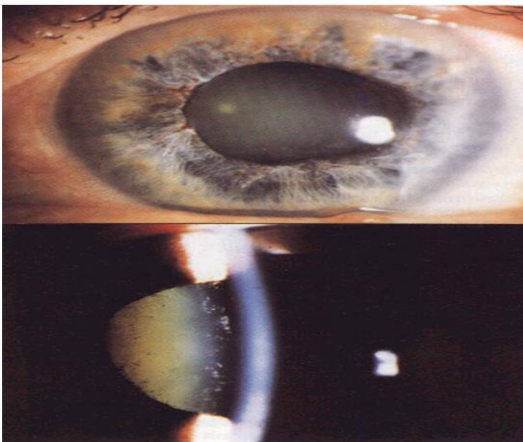
- In **acute angle closure glaucoma**, there is an abrupt increase in pressure and the eye becomes photophobic...painful due to ischemia...watering of the eye...loss of vision..
- Pt feels unwell...nausea...abdominal pain
- On exam.: red eye...visual acuity is reduced...cloudy cornea...pupil is oval fixed and dilated

Acute congestive angle-closure glaucoma

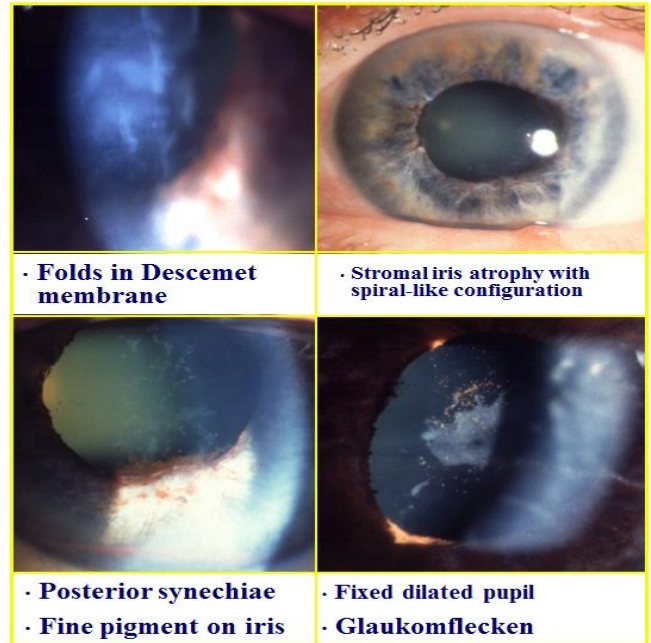
Signs



Post-congestive angle-closure glaucoma



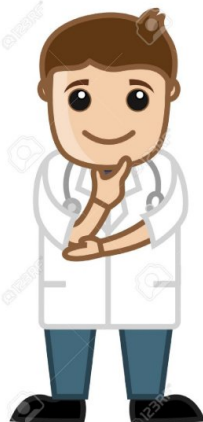
Signs Of Postcongestive Angle-Closure Glaucoma



Treatment of Acute Congestive Angle-Closure Glaucoma

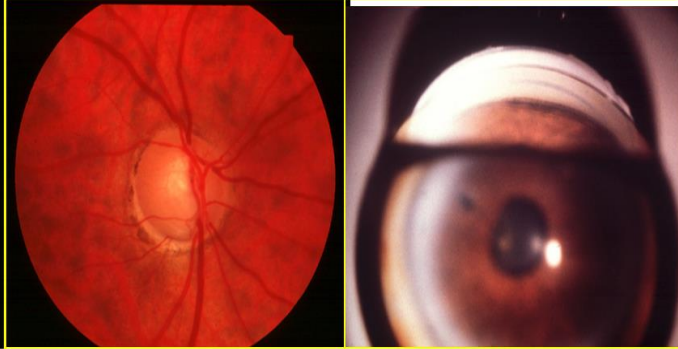
1. Acetazolamide 500 mg i.v.
2. Hyperosmotic agents - if appropriate
 - Oral glycerol 1-1.5 g/kg of 50% solution in lemon juice
 - Intravenous mannitol 2g/kg of 20% solution
3. Topical therapy
 - Pilocarpine 2% to both eyes
 - Beta-blockers
 - Steroids
4. YAG laser Iridotomy
 - To both eyes when cornea is clear

اللجنة العلمية Group C



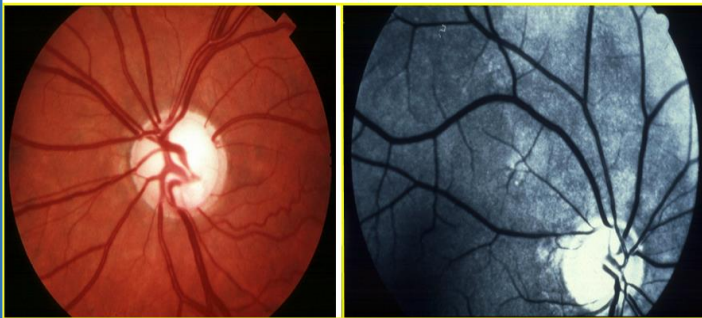
Chronic angle-closure glaucoma

Signs



- Similar to POAG with cupping and field loss
- Easily missed unless routine gonioscopy performed
- Variable amount of angle closure

End-stage damage



- All neural disc tissue is destroyed
- Disc is white and deeply excavated
- Atrophy of all retinal nerve fibres
- Striations are absent
- Blood vessels appear dark and sharply defined

Treatment

- Acute stage
 1. Mannitol (IV), Diamox (IV/oral), Glycerine (oral), Pilocarpine, Beta Blockers
 2. Yag or Surgical Peripheral Iridectomy (PI)
 3. Trabeculectomy (if above fails)
- Chronic stage
 1. Trabeculectomy
- Absolute Stage
 1. Retrobulbar Alcohol Injection
 2. Cyclo-cryotherapy
 3. Enucleation

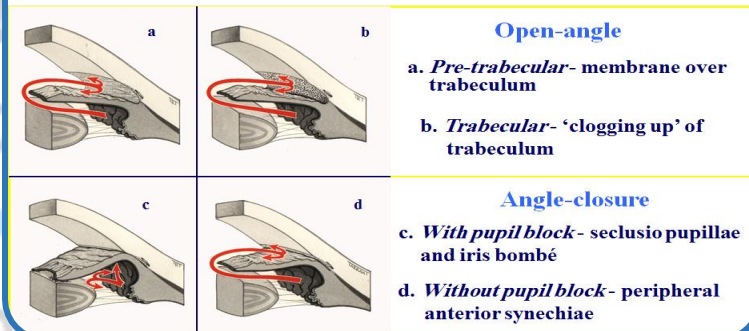
Secondary Glaucoma

Secondary Glaucoma

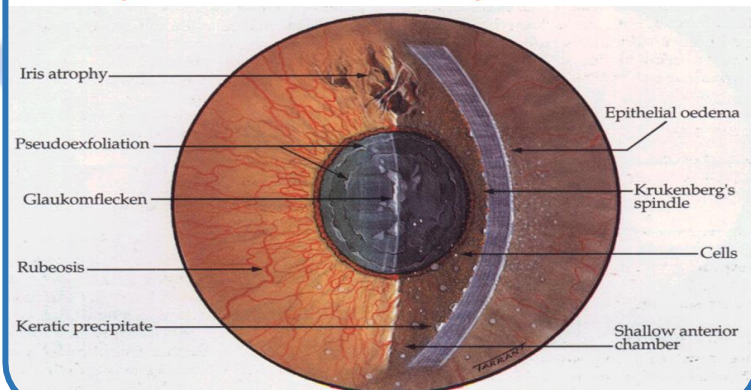
(Rise In IOP Secondary To A Disease In The Eye)

- Corneal Causes (Corneal ulcer, leucoma adherent)
- Anterior Chamber (Hyphaema, lens dislocation)
- Iris (Iridocyclitis, Rubeosis Iridis)
- Lens (phakomorphic glaucoma, phakolytic glaucoma, Anterior dislocation of lens, lens matter post surgery, pseudoexfoliation syndrome)
- Haemorrhagic Glaucoma (due to vitreous hge.)
- Neovascular Glaucoma (DM and CRVO)
- Intraocular Tumors
- Drugs (Steroid)
- Much rarer than the primary glaucoma
- Sign and symptoms depends on the rate at which the IO pressure rises...mostly are symptomless
- TTT: same as 1ry + treat the underlying cause
 - In severe cases: laser or cryoprobe are used to ablate the ciliary processes

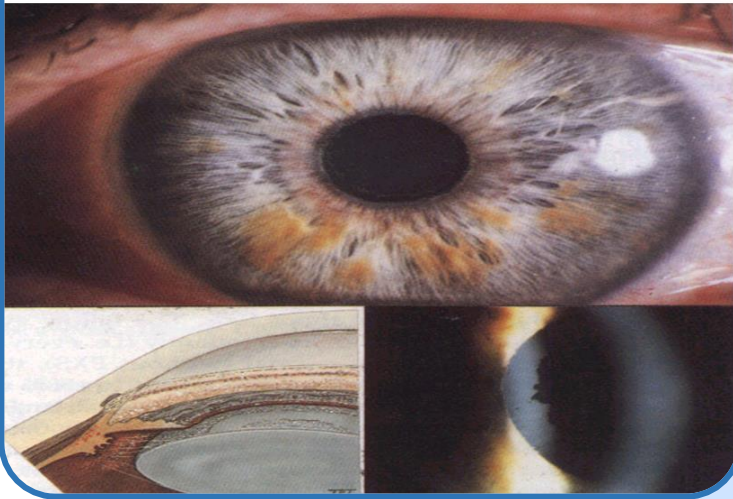
Classification of Secondary Glaucomas



Possible Findings On Slitlamp Biomicroscopy In Eyes With Various Secondary Glaucomas

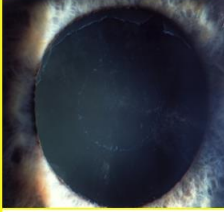
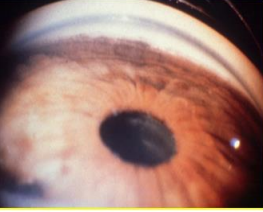


Pseudoexfoliation Syndrome



Pseudoexfoliation Glaucoma

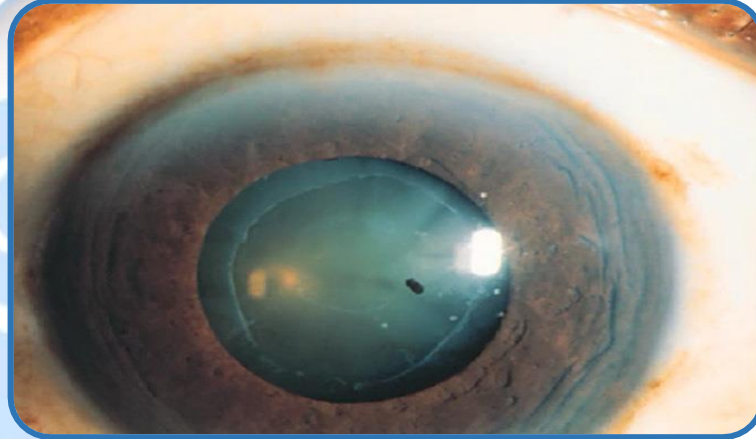
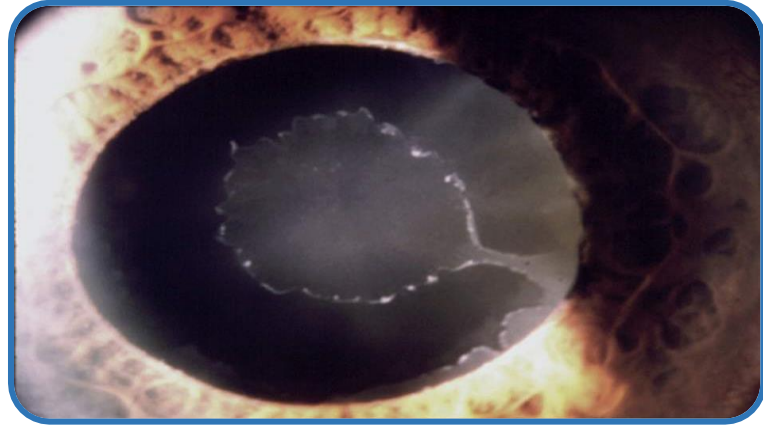
- Secondary trabecular block open-angle glaucoma
- Affects elderly, unilateral in 60%
- Prognosis less good than in POAG

Pseudoexfoliative material	Iris sphincter atrophy	Gonioscopy
		
Central disc with peripheral band	On retroillumination	Trabecular hyperpigmentation - may extend anteriorly (Sampaolesi line)

Pseudoexfoliation Syndrome



Deposition of abnormal fibrillary material on different structures of the anterior ocular segment. Frequent cause of secondary open angle glaucoma

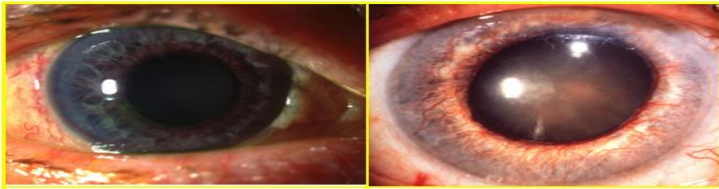


Rubeosis Iridis

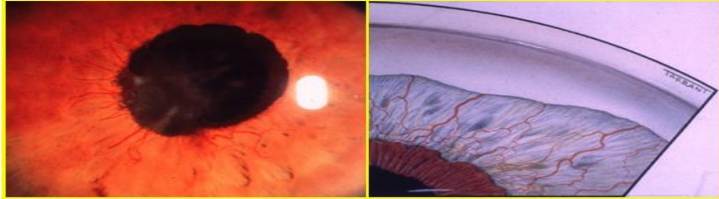


Deposition of abnormal fibrillary material on different structures of the anterior ocular segment. Frequent cause of secondary open angle glaucoma

Signs of advanced neovascular glaucoma

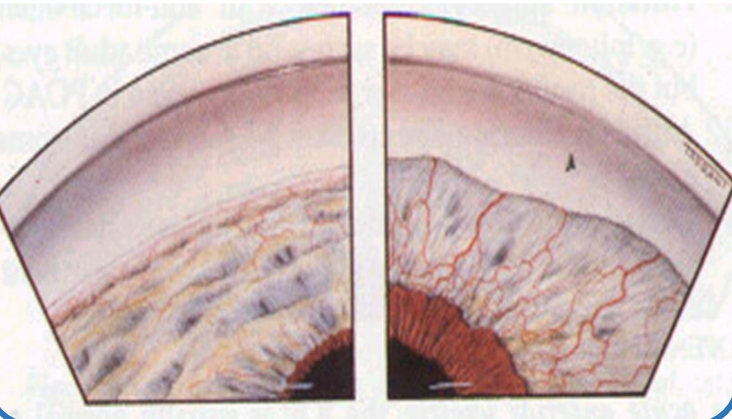


Severely reduced visual acuity, congestion and pain Severe rubeosis iridis

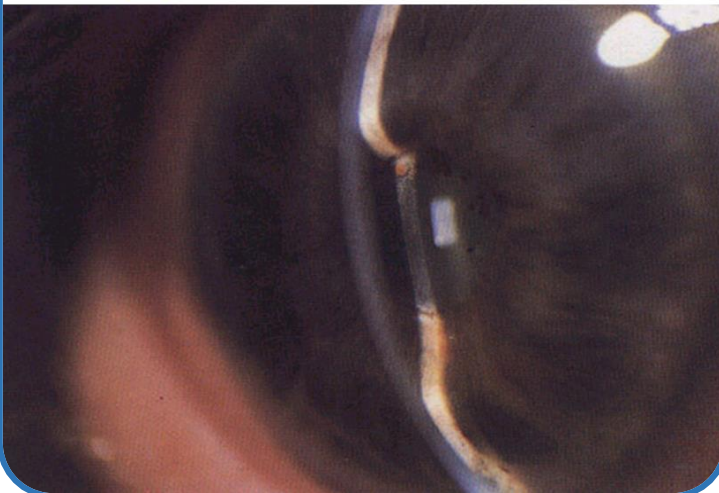


Distortion of pupil and ectropion uveae Synechial angle closure

Angle in Neovascular glaucoma

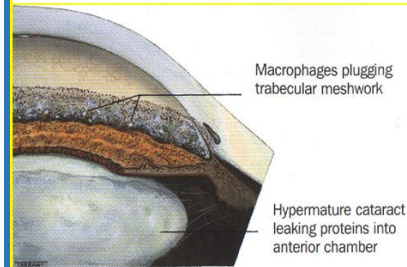


Secondary angle-closure glaucoma caused by pupil block in chronic iridocyclitis

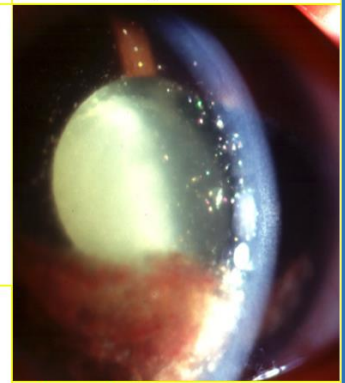


Phacolytic glaucoma

Pathogenesis



Signs

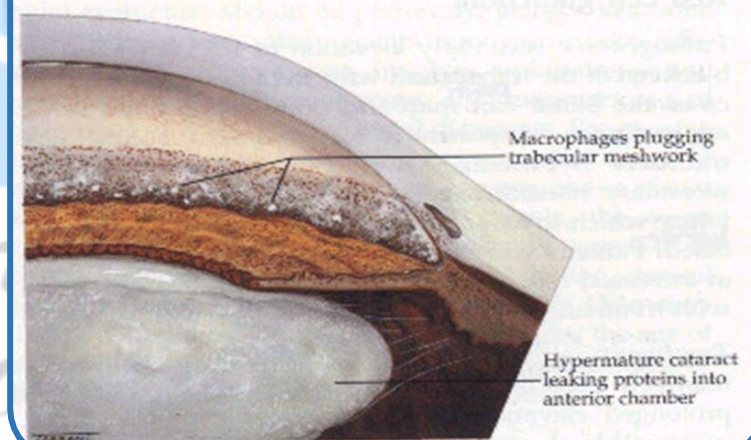


Treatment

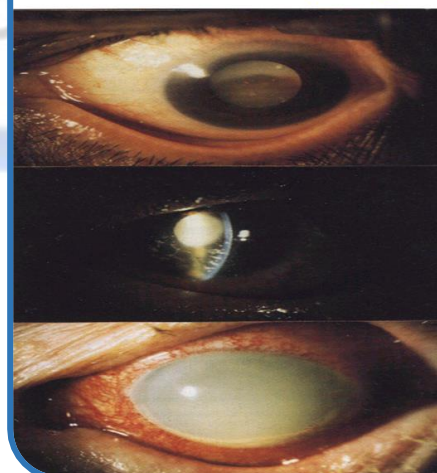
- Control IOP medically
- Remove cataract

- Deep anterior chamber
- Floating white particles

Pathogenesis of Phacolytic Glaucoma



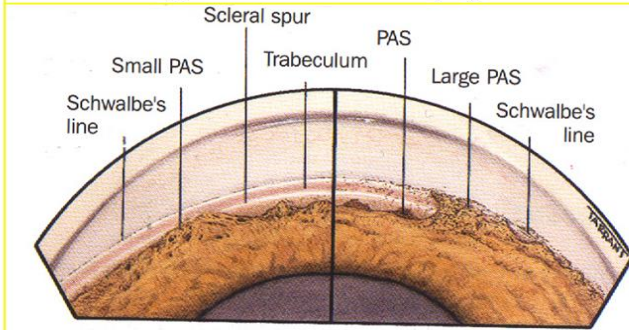
Phacolytic glaucoma



- Morgagnian cataract
- floating white particles in aqueous
- pseudohypopyon

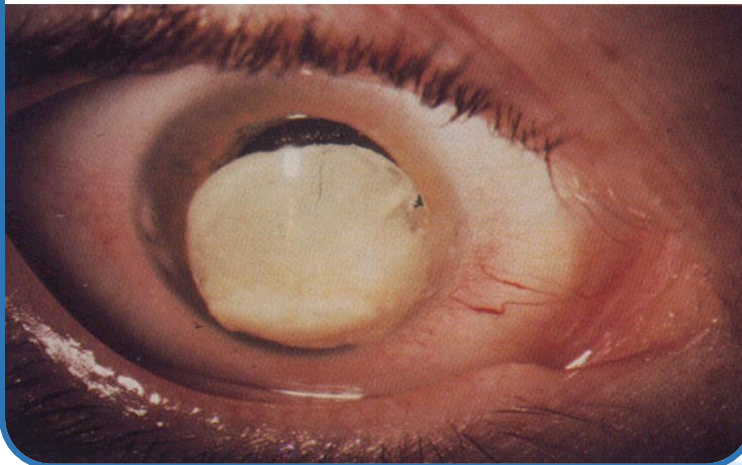
Inflammatory glaucomas

Angle-closure without pupil block

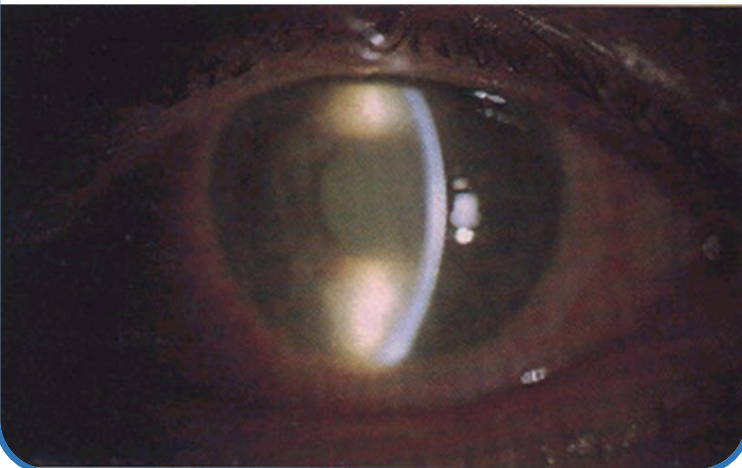


- Caused by progressive synechial angle closure
- Anterior chamber is deep

Mature cataract dislocated into anterior chamber



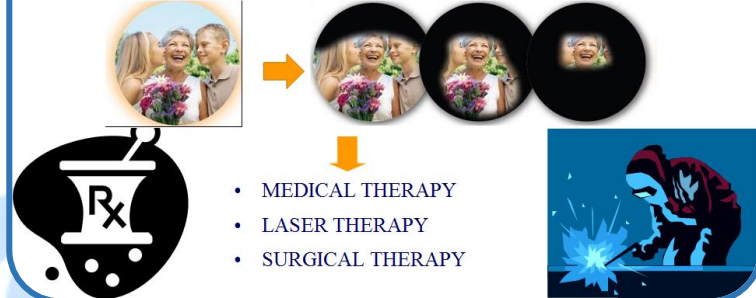
Ghost cells in the aqueous



Treatment of Glaucoma

GENERAL TREATMENT OPTIONS FOR GLAUCOMA

THE GOAL OF GLAUCOMA TREATMENT IS REDUCE THE PRESSURE BEFORE IT CAUSES GLAUCOMATOUS LOSS OF VISION



GLAUCOMA Treatment

Medical

- ❖ Miotics
- ❖ Beta-blockers
- ❖ Carbonic anhydrase inhibitors
- ❖ Prostaglandin analogues
- ❖ Alpha-2 agonists

Surgical

- ❖ Argon laser trabeculoplasty
- ❖ Trabeculectomy
- ❖ Filtering procedure
- ❖ Cyclocryotherapy
- ❖ Cyclolaser ablation
- ❖ Iridotomy

MEDICAL THERAPY

AQUEOUS SUPPRESSANTS

- **ADRENERGIC ANTAGONISTS (BETA BLOCKERS)**
 - NONSELECTIVE
 - TIMOLOL, LEVOBUNOLOL, CARTEOLOL (ISA+), METIPRANOLOL
 - SELECTIVE
 - BETAXOLOL
- **ADRENERGIC AGONISTS (SELECTIVE ALPHA-2 AGONISTS)**
 - APRACLOPIDINE
 - BRIMONIDINE
- **CARBONIC ANHYDRASE INHIBITORS**
 - SYSTEMIC
 - ACETOSOLAMIDE
 - TOPICAL
 - DORZOLAMIDE
 - BRINZOLAMIDE

OUTFLOW FACILITATIVE DRUGS

- **CHOLINERGICS**
 - PILOCARPINE
- **PROSTAGLANDINS**
 - LATANOPROST
 - TRAVOPROST
 - BIMATOPROST

FIXED COMBINATIONS

TIMOLOL MALEAT

+

Dorzolamide Latanoprost Travoprost
↓ ↓ ↓
COSOPT XALACOM DOUVRAT

• BRINZOLAMIDE
• DORZOLAMIDE
• TOPICAL
• ACETOSOLAMIDE
• SYSTEMIC

COSOPT XALACOM DOUVRAT
↓ ↓ ↓
Dorzolamide Latanoprost Travoprost

Drugs to treat glaucoma

1. Beta blockers
2. Sympathomimetics
3. Miotics
4. Prostaglandin analogues
5. Carbonic Anhydrase inhibitors
 - Topical
 - Systemic

- The initial therapy is usually medical, except in advanced cases. The chosen drug should be used at its lowest concentration, as infrequently as possible, to achieve the desired effect. If possible, the drug with the fewest potential side effects should be chosen. Initial treatment is usually with either a β -blocker or a sympathomimetic

Antiglaucoma Drugs

β -Blockers

- All β -blockers reduce IOP by decreasing aqueous secretion

Systemic Side Effects

1-Pulmonary side effects in the form of bronchospasm may be induced.

2- Cardiovascular effects such as bradycardia and hypotension can result from the β_1 -adrenergic blocking action. Delayed recovery from hypoglycaemia. In general the use of β -blockers in diabetic patients is not, however, contraindicated, although betaxolol may be preferred because it does not impair glycogenolysis..

Others as fatigue, depression, confusion, hallucinations, headache, nausea, dizziness,

- PREPARATIONS
- Timolol (0.25% and 0.5%)
- Betaxolol 0.5%
- Levobunolol 0.5
- Carteolol

Sympathomimetic

Mode Of Action

- Sympathomimetics are α - and β -adrenergic agonists. They increase aqueous outflow through their α -agonist action and decrease aqueous inflow through their α -agonist activity.

Preparations

- Adrenaline 0.5%, 1% and 2%

- The onset of action is within 1 hour and the duration is up to 12-24 hours; administration is twice daily. Dipivefrin (Propine) 0.1%

Miotics

Mode Of Action

- In POAG miotics are thought to reduce IOP by inducing contraction of the longitudinal muscle of the ciliary body. This pulls on the scleral spur and produces changes in the trabeculum which enhance aqueous outflow .

Local Side Effects

- In contrast to systemic side effects, local side effects may be considerable and are responsible for low compliance.
- Miosis may cause impairment of night vision, reduced visual acuity in the presence of axial lens opacities, generalized constriction of the visual field and an apparent increase in the size of visual field defects.
- Spasm of accommodation may cause myopia Retinal detachment
- Increased permeability of the blood-aqueous barrier is undesirable because it allows the transfer of proteins, fibrin and cells into the aqueous humour.

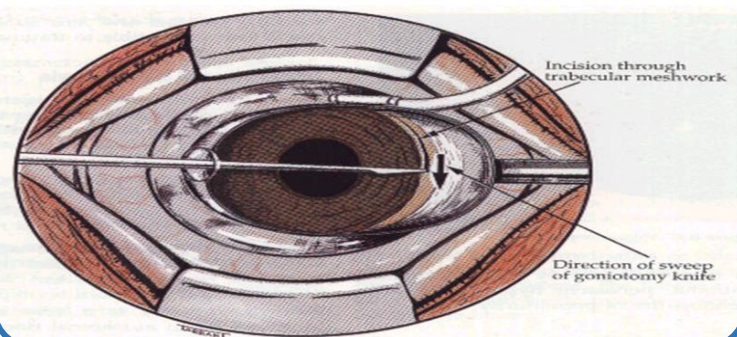
Preparations

- Pilocarpine 1%, 2%, 3% and 4%

Surgeries for Glaucoma

- Goniotomy
- Trabeculotomy
- Trabeculectomy
- Trabeculectomy + Antimetabolites
- Surgical Peripheral Iridectomy (PI)
- Cyclocryotherapy
- Laser
 - Yag Peripheral Iridotomy (YAG PI)
 - Argon Laser Trabeculoplasty (ALT)

Goniotomy



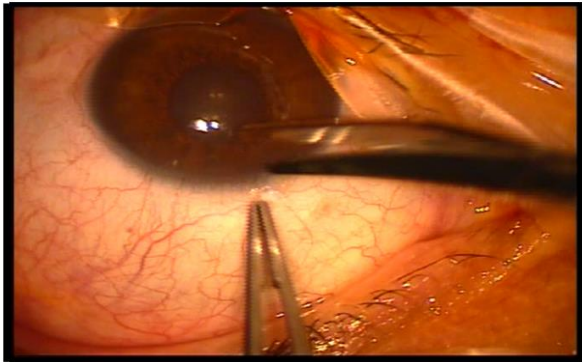
Indications for Trabeculectomy

1. Failed medical therapy and laser trabeculoplasty
2. Lack of suitability for trabeculoplasty
 - Poor patient co-operation
 - Inability to adequately visualize trabeculum
3. As primary therapy in advanced disease

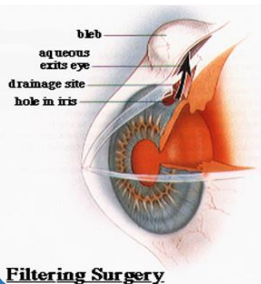
Trabeculotomy



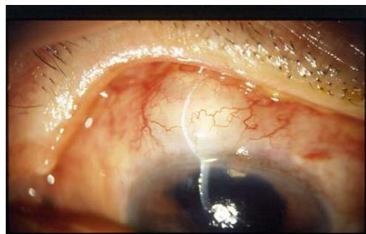
TRABECULECTOMY



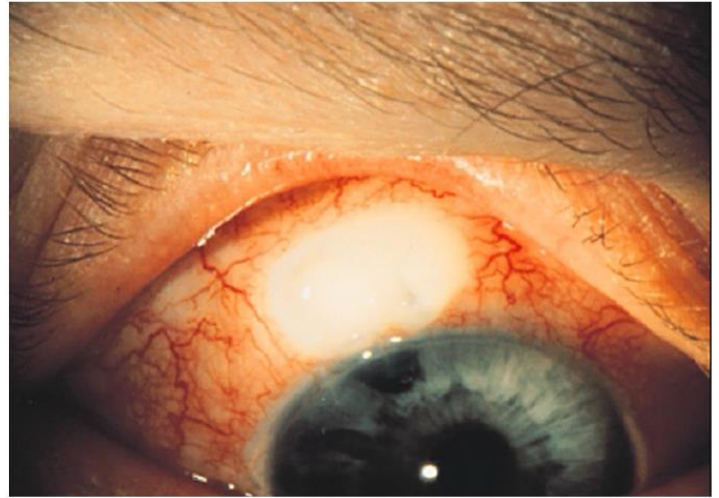
GLAUCOMA Filtration blebs



Filtering Surgery



Bleb after trabeculectomy



Filtration blebs



Type 1

- Thin and polycystic
- Good filtration



Type 2

- Flat, thin and diffuse
- Relatively avascular
- Microcysts present
- Good filtration



Type 3

- Flat
- Engorged surface vessels
- No microcysts
- No filtration



Encapsulated

- Localized, firm cyst
- Engorged surface vessels
- No filtration

Complications of surgery:

- 1- swallowing of the AC in the immediate postop. Period (cause damage to the lens and cornea)
- 2- IO infection
- 3- accelerated cataract development
- 4- failure to reduce intraocular pressure adequately
- 5- an excessively low pressure (hypotony) which may cause a macular edema

Late bleb infection

Predispositions

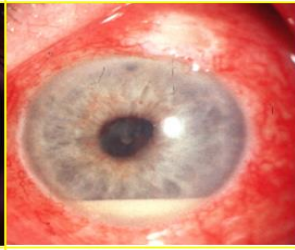
- Thin-walled, cystic bleb
- Use of adjunctive antimetabolites
- Bleb trauma

Blebitis



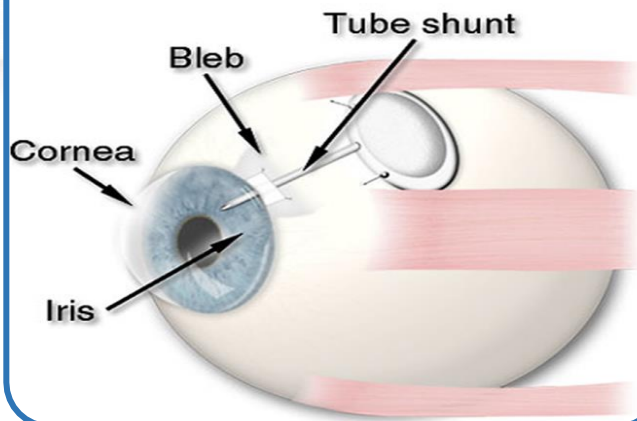
- Subacute onset
- Milky bleb
- No hypopyon
- Good prognosis

Endophthalmitis

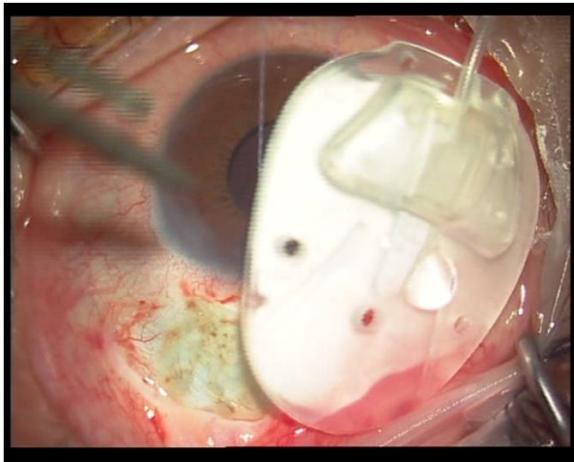


- Acute onset
- Hypopyon
- Guarded prognosis

Glaucoma Tube Shunt Implantation



AHMED GLAUCOMA VALVE



Surgeries for Glaucoma

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- Trabeculectomy + Antimetabolites
- Surgical Peripheral Iridectomy (PI)
- Cyclocryotherapy
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Laser Therapy

- Laser trabeculoplasty
 - Argon laser trabeculoplasty (argon laser)
 - Selective laser trabeculoplasty (nd:yag)
- Cyclophotocoagulation
 - Transscleral (nd:yag, diode)
 - Transpupillary (argon)
 - Transvitreal (during vitrectomy)
 - Endoscopic (argon)



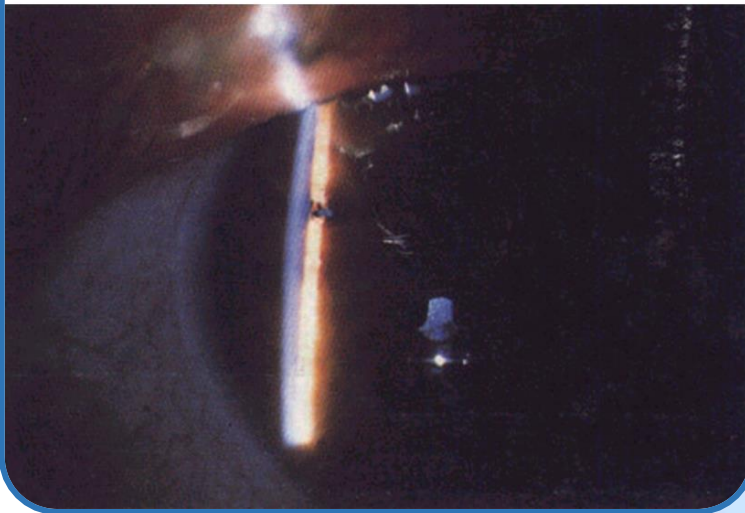
ARGON LASER
TRABECULOPLASTY



DIODE LASER TRANSSCLERAL
CYCLOPHOTOCOAGULATION

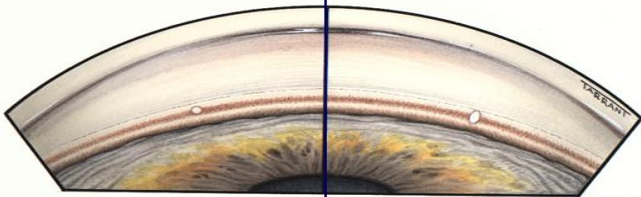
CYCLOPHOTOCOAGULATION
DIODE LASER TRANSSCLERAL

Appearance following YAG laser PI



Laser trabeculoplasty

- Failed medical therapy
- Primary therapy in non-compliant patients



- Application of 50-100 burns to junction of pigmented and non-pigmented trabeculum
- Correct focus with round aiming beam

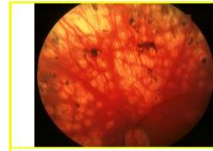
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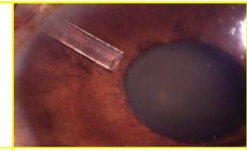
Treatment Options Of Neovascular Glaucoma

Topical

- Atropine and steroids to decrease inflammation
- Beta-blockers



Panretinal photocoagulation
- in early cases



Artificial filtering devices
- in very advanced cases



Cyclodestructive procedures
- to relieve pain



Retrobulbar alcohol injection
- to relieve pain

DIODE LASER CYCLOPHOTOCOAGULATION



3- Cyclodestructive procedures

- Cyclodestructive procedures lower IOP by destroying part of the secretory ciliary epithelium. The main indication is intractable glaucoma, usually associated with permanent synechial angle closure, which has failed to respond to other measures aimed at increasing aqueous outflow. The two main types are cyclocryotherapy and YAG laser cycloablation.

Cyclocryotherapy



